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|-----------|----------|-------------|------------------------------|---|
| 1% | 2% | 1:40,000 | 33% | 3 times |
| | | 1:40,000 | 90% | |
| 1% | 2% | 1:60,000 | 22% | 3½ times |
| | | 1:60,000 | 74% | |
| 1% | 2% | 1:80,000 | 10% | 7 times |
| | | 1:80,000 | 69% | |

"Anesthesiology" July 1942, page 408, table 10.



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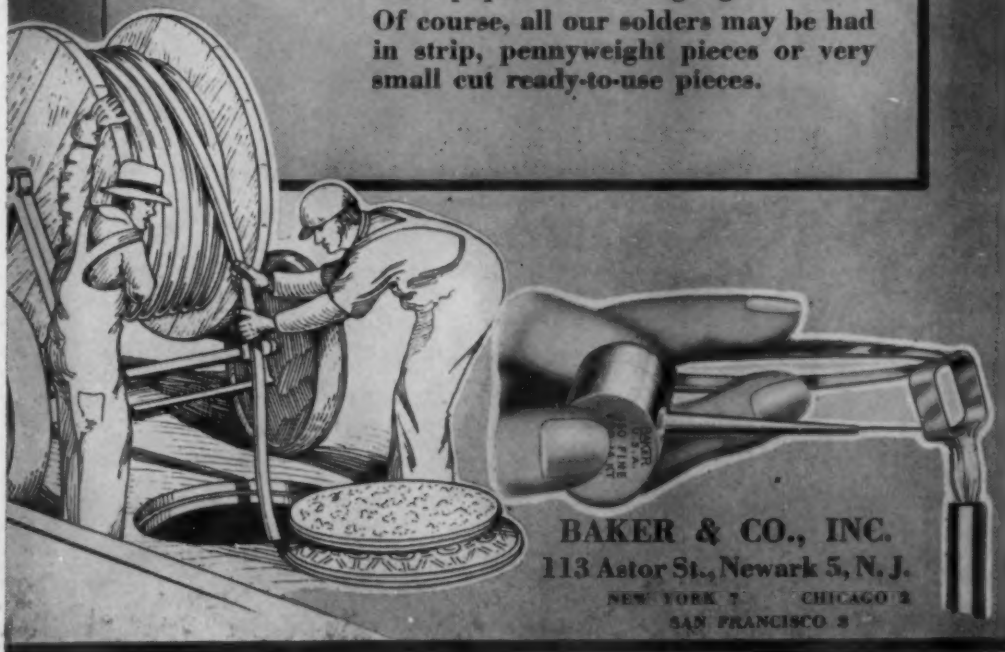


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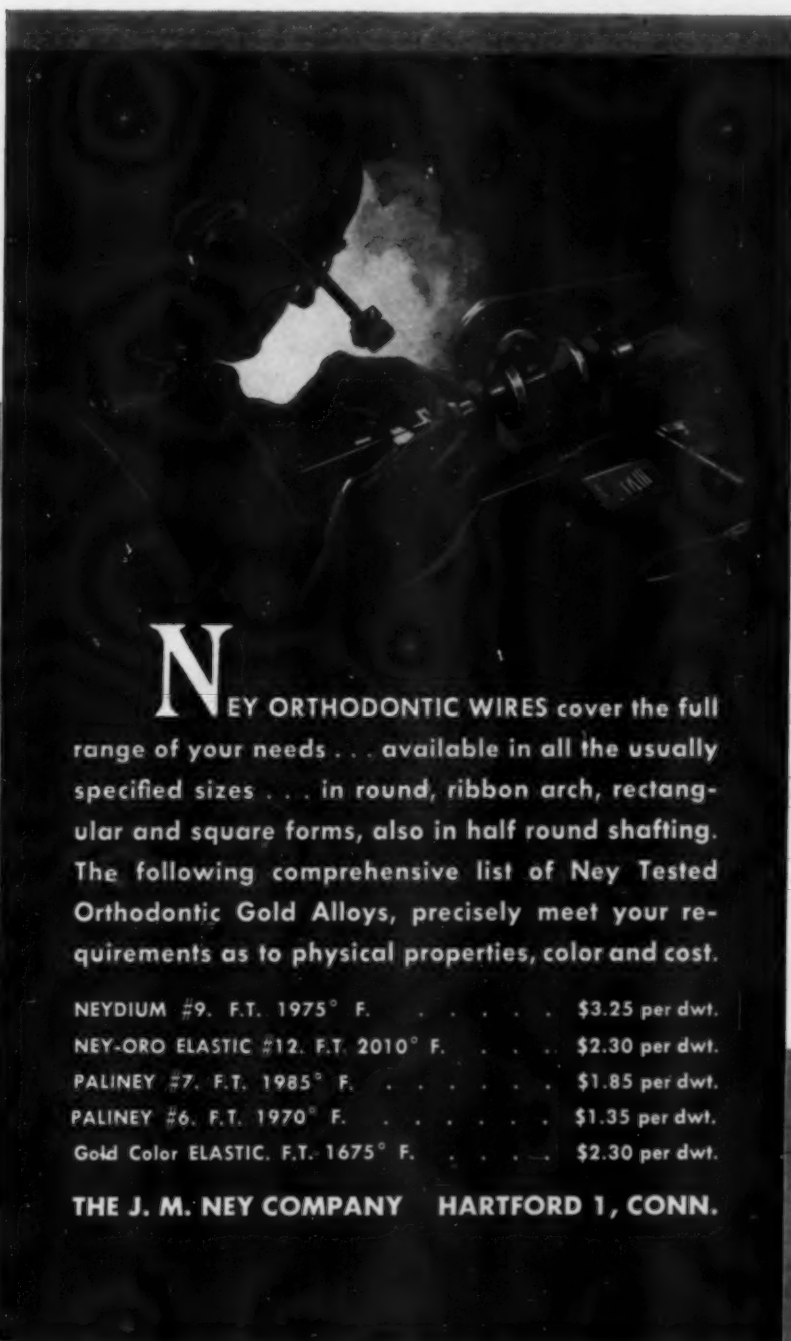
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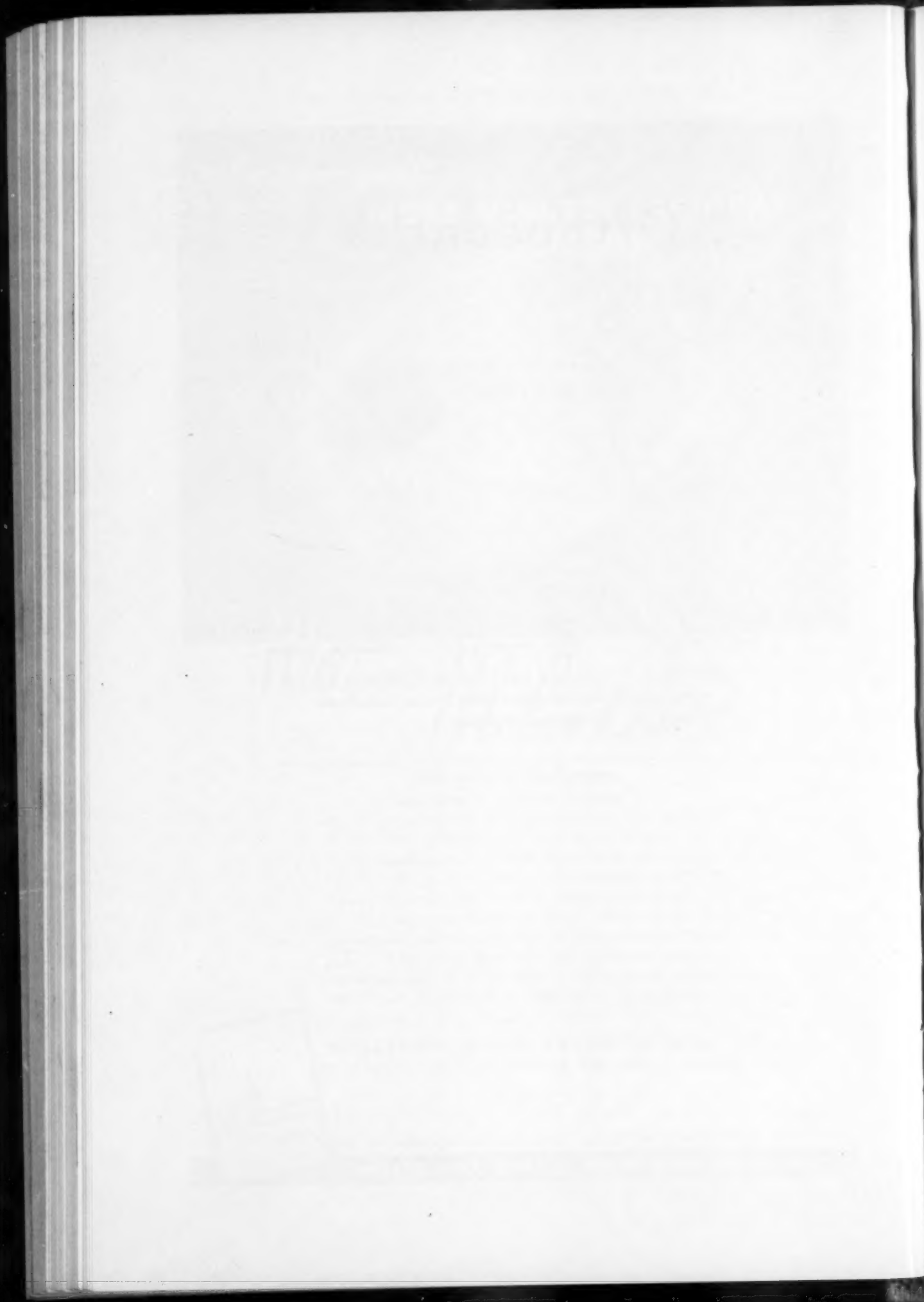
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Original Articles

OCCIPITAL AND CERVICAL ANCHORAGE AND THEIR APPLICATION TO THE ORTHODONTIC PROBLEM

HARRY E. JERROLD, D.D.S., BROOKLYN, N. Y.

IT WOULD be proper to fix in our minds what anchorage means, before discussing occipital and cervical anchorage.

McCoy states, "Anchorage consists in the selection of adequate and properly distributed resistance units for the control and direction of force." In orthodontics, the applied force is exerted by the mechanism on the teeth that are in malocclusion. In turn, there is a reaction that must be resisted. The anchorage must be strong enough to withstand this applied force.

Atkinson claims that there is no such thing as stationary anchorage, but we can use a maximum of resistance units, pitted against a minimum of response units, if we employ strategy of a regional and rational type.

There are two sources of anchorage available: extraoral and intraoral. The essayist will present the extraoral, or what is commonly known as occipital and cervical anchorage, for your consideration.

The occipital and cervical anchorage was among the first to be used in orthodontic practice. The early practitioners recognized the advantage of locating the base of anchorage completely outside the immediate field of action. As means develop for applying this strategy in a scientific and skillful manner, in all probability it will be considered more and more an indispensable base from which force is derived for the movement of teeth.

This can only be accomplished by a full appreciation of dentofacial relations, and the adoption of applicable variations in methods and apparatus which will make the proper corrective movements possible.

As far back as 1822, Gunnell claimed to have used occipital anchorage, but he did not describe its use until 1841. In the meantime, Kneisel published this idea of anchorage in 1836. Kingsley in 1875, Angle in 1889, and others used occipital anchorage to a great extent. During the period that followed, however, this type of anchorage gradually fell into disfavor.

One of the greatest objections to the more general adoption of the occipital force has been the discomfort and irritation, if not actual pain, which the various forms of apparatus that were employed by the profession gave to many patients,

and which so frequently caused them to omit wearing it a sufficient portion of the time to be of real service.

The author communicated with Dr. Carl B. Case, of Milwaukee, Wisconsin, and received a copy of a paper read before the American Society of Orthodontists, entitled "Occipital and Cervical Anchorage." It was in 1910 that Dr. Case introduced the method of employing the neck or the base of the skull to sustain at least part of the reactive force and termed it "cervical or cervico-vertebral anchorage." He states, "It is obvious that this apparatus should undoubtedly prove efficient in all cases of dental mal-position, where a horizontal direction of pull sustained by an anchorage remote from the teeth is considered an advantage." Dr. Case describes in detail the merits of cervical and occipital anchorage in this paper and it should be read by all who wish to employ this form of anchorage.

An occipital apparatus should be one that can be perfectly fitted by the operator to the form and requirements of the individual patient, with no projecting portions to interfere with the patient while at rest, and one which can be easily adjusted by the patient and worn with the least possible discomfort during sleeping hours. The principal direction of its movement is upward and backward, with a tendency toward the production of a movement, when applied to the teeth, that is frequently demanded and which cannot be accomplished more efficiently in any other way.

There were many types of cumbersome headgear and auxiliary apparatus which were used in the early days. Calvin Case describes a head cap made of thin metallic ribbons which were properly shaped and provided with adjustable gears for fitting it to the side of the head. He claimed that it could be adjusted to lie smoothly upon the surface and place the force where it was least felt, leaving the head almost entirely free. Silk elastics of the proper gauge or thickness were used for the motive power. They were buttoned to the head cap with glove fastener attachments and passed through lock swivel loops at the end of the dental bow to sliding buckles for adjusting the amount of force. All metal parts were highly nickel plated. (Fig. 1.)

The use and effectiveness of the apparatus depend largely upon the manner in which the several parts are adjusted and fitted. The head cap now in use by the writer consists of a cotton hair net of close mesh, which is made in three different sizes, to fit the head snugly. It covers the ears and is tied by two strings, fastening below the chin by means of a bowknot. A piece of grosgrain ribbon, $1\frac{1}{2}$ inch wide, is placed in the center of the cap, from front to back, and stitched down on both sides. This merely acts as a reinforcement. Another piece of ribbon, of similar width, is placed on either side of the lower and back part of the cap, from the center to the front, and is either stapled or sewn on by hand or a sewing machine, the last method being most desirable. Two additional pieces of ribbon are placed right above these, slightly overlapping them, and stitched down. Their purpose is for additional reinforcement and to afford a surface to which a hook may then be sewn, on each side of the cap, at the proper horizontal plane for the reception of elastics. (Fig. 2.)

When this type of cap is properly fitted, we have a headgear that rests well back on the head, does not interfere with the ears and face, and apparently is not uncomfortable, since very few children or adults register any objections to its nightly use.

The fitted head cap described above is merely a potential unit of resistance and can be called upon to act only when it is harnessed with auxiliary types of mechanism. The appliance is only an instrument having certain necessary

requirements, which are described by Atkinson as follows: "It is our concern to select orthodontic appliances capable of delivering to the teeth and jaws the most nearly correct amount of controlled force capable of inciting proper cellular activity in a selected area of the anatomy of a particular individual." It is now known that when the slightest amount of artificial force is applied to a tooth, it will incite cellular activity, causing the tooth to move in the direction of the applied force, provided the force is not too great.

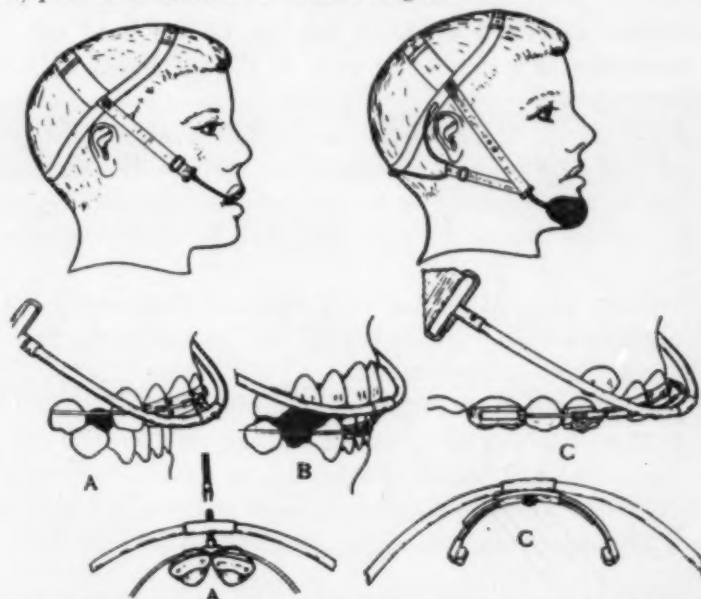


Fig. 1.—(From Case, Calvin: Dental Orthopedia, ed. 2, 1921, C. S. Case Co.)



Fig. 2.—(Taken by Dr. Ralph Waldron.)

It is the opinion of the writer that the occipital and cervical anchorage meets with the specifications of an efficient mechanism, and he deems it advisable for all who practice our profession to thoroughly acquaint themselves with the principles and latest methods of its application.

The writer became acquainted with this type of strategy while attending the fourth orthodontic conference at the University of Southern California, in

August, 1940. The lectures and demonstrations of Drs. Albin Oppenheim and Spencer R. Atkinson were a source of inspiration which aroused the interest of the essayist to the possibilities of occipital and cervical anchorage in modern orthodontic practice. Its applications are many and varied, and may be divided into the following three major categories: (1) Stabilizing agent. (2) Active unit of force. (3) Means for retention.

In the January, 1941, issue of the *Angle Orthodontists*, Dr. Charles Tweed of Tucson, Arizona, describes in detail the use of the head cap for occipital and cervical anchorage as a stabilizing unit, in the treatment of Angle Class II, Division 1 malocclusion.

Dr. Samuel J. Lewis describes the use of a headgear to be worn at night during the period of anchorage preparation in the mandibular arch. He says, "In this technique, the headgear is purely for the purpose of assisting in the preparation of mandibular anchorage, and not to move the maxillary teeth distal."

Drs. Oppenheim and Atkinson, of Pasadena, California, have employed the headgear apparatus for both the distal and mesial movement of teeth, in Angle Class II and Class III malocclusions.

Occipital anchorage for retention was employed by Dr. Calvin Case and described in his *Dental Orthopedia*, second edition, published in 1921.

The use of occipital and cervical anchorage, as a means of retention, was also presented by Dr. Ralph Waldron in New Orleans at the meeting of the American Association of Orthodontists and Inter-American Orthodontic Congress in March, 1942.

The writer has employed occipital and cervical anchorage for each of the three forms of strategy previously mentioned, with a great deal of satisfaction.

In any form of malocclusion where you wish to relieve the strain upon a unit of resistance, or to reinforce that unit, the occipital and cervical anchorage may be employed to good advantage.

In Angle Class II, Division 1, malocclusion, where the mandibular teeth are in abnormal axial inclination, the following type of headgear apparatus has been used successfully as a stabilizing unit. There are two methods of assemblage: (1) Ribbon elastics are sewn to the head cap with suitable traction hooks which engage intermaxillary hooks on the mandibular labial arch. (2) Tubes of 0.041 or 0.042 inch inside diameter, $\frac{3}{8}$ inch long, are soldered to the molar bands, as close to the gingival third of the band as the soft tissue will permit. A 0.040 steel arch wire, made to conform to the mandibular arch, is inserted into these tubes, making certain that there is no frictional resistance. A scratch mark is made at the median line to enable the arch to be replaced in its proper position.

A 0.050 steel wire is conformed to the anterior segment to the arch labially, terminating mesial to the canines. At this point, it is bent outward for about $\frac{1}{2}$ inch and then continued backward to the molar region; there it is formed into a hook. The outer section is then soldered to the arch, at its anterior segment. This complete arch is then replaced into the tubes and any excess length removed. (Fig. 3.)

Commercial No. 12 rubber bands are used in this application, care being exercised that the arch fits snugly against the labial surfaces of the mandibular anterior teeth.

When the stabilization of the molars, in conjunction with the anterior segment, is desired, stops may be soldered to the arch, anterior to the buccal tubes.

The apparatus, for use as a direct unit of force, is constructed as follows: Molar bands are fitted to the maxillary first molars, to which are either soldered

or spot-welded flanged buccal tubes, as close to the gingiva as possible. These buccal tubes are 0.041 or 0.042 inch (inside dimension); large enough to receive an arch 0.040 inch in diameter, without binding. Immediately below these tubes there is, as a rule, sufficient space for the attachment of either a rectangular buccal sheath or any other type of molar band attachment.

The type first used by the essayist, designed by Dr. Oppenheim, consisted of a labial arch 0.040 inch in diameter, which was fitted to conform to the maxillary teeth and was properly aligned to the plane of the gingival third of the crowns of the maxillary teeth.

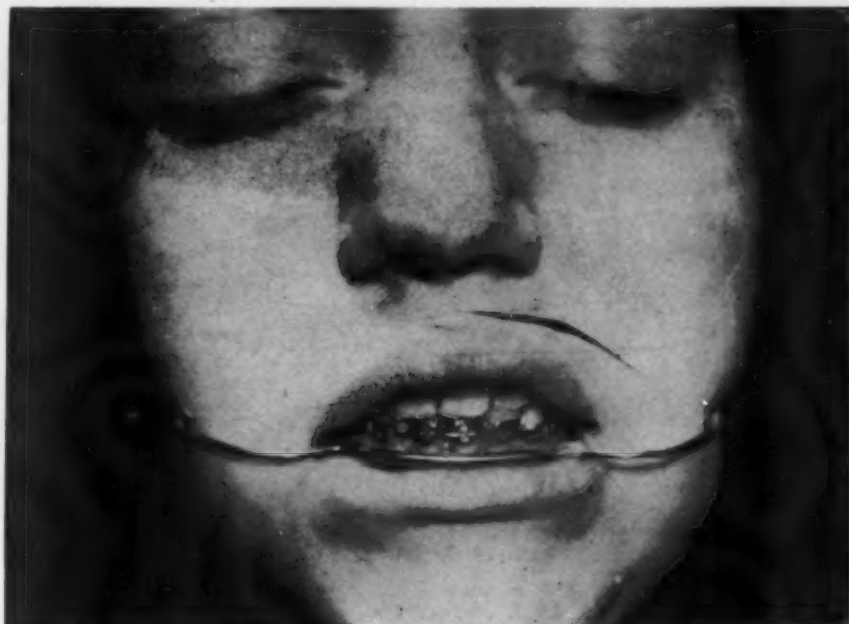


Fig. 3.—Showing the application of occipital arch, in conjunction with edgewise mechanism in the mandible.

The center of the arch was marked and a small piece of 0.040 wire soldered to it so that it projected forward about 5 millimeters. The arch was then inserted into the buccal tubes and marked for the soldering of suitable stops, anterior to the buccal tubes. The arch bow had a receptacle in the center which received the projection on the labial arch. The ends were curved to receive the elastics, which may be either the cloth type, with suitable attachments, or the regular rubber bands. This is worn at night only, giving the teeth the necessary rest period between the applications of force, as advocated by Oppenheim, and is very efficient in moving the molars distally. The interdental fibers connecting each tooth cause the premolars to follow in the same direction. Interdental or transseptal fibers are defined by the late Rudolf Kronfeld as follows: "These fibers are confined to the area between two adjacent teeth; they run from the cementum at the neck of one tooth in a more or less horizontal direction across the crest of the interdental septum to the cementum of the next tooth. The arrangement of the transseptal fibers indicates that their functions consists in maintaining the mesiodistal relationship between neighboring teeth and in stabilizing the teeth against separating forces."

During the distal movement of the buccal segments, the anterior segment usually follows due to these factors: (1) The pressure which originally displaced them has been relieved; (2) the natural drift of incisors, cuspids, and premolars is toward the distal, aided by lip pressure and the action of the buccinator muscle.

The writer is now using an arch designed by Dr. Ralph Waldron for distal movement of maxillary teeth. This improved arch, as previously described, eliminates the necessity of excess equipment, is considerably simpler in its application, and is more readily tolerated by the patient. Its description bears repetition. It consists of a labial arch of 0.040 steel (or other suitable material) which is shaped for each individual. To this, another anterior segment of 0.050 steel with wings is soldered and is so shaped that it clears the lips and cheeks and is curved at both ends to receive the elastics. (Fig. 4.)

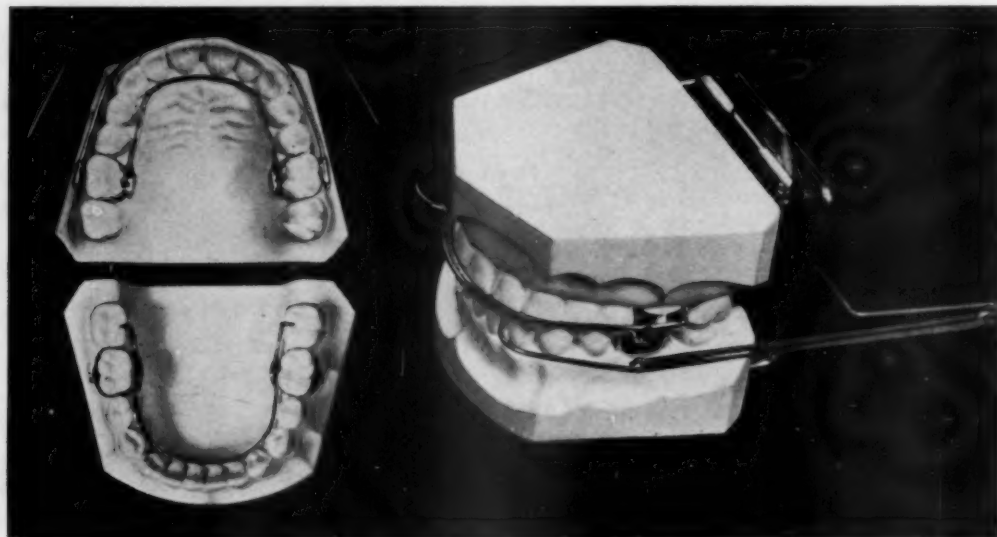


Fig. 4.—Lingual arches for stabilization as well as labial arch for use with headgear.

No. 12 elastic bands are used, one on each side, each exerting 6 to 8 ounces of force. These are replaced every week. If more force is desired, two elastics may be worn on each side. One must be mindful, however, that too much force is contraindicated and may have a tendency to open the bite. This type of apparatus does not interfere with sleep or rest. It is advisable to mention, at this time, that the anterior segment of the arch should not be more than 1 mm. away from the labial surface of the maxillary incisors.

In a case where distal movement of only one lateral segment is desired, as in unilateral cases, the essayist solders stops on both sides of the labial arch in front of the buccal tubes, and employs two elastics on the side to be moved distally and only one on the side that is to remain passive.

Occasionally, the stop is eliminated on the normal side and soldered only to the side of the arch to be moved, using two elastics for the active side and one on the side where no movement is desired.

In Angle Class I malocclusion, where the mandibular arch is normal mesio-distally, in relation to skull anatomy, and the maxillary buccal segments have drifted forward, it is the practice of the essayist to move the maxillary second molars distally by the use of a span glider or sliding yoke, sliding on either an Angle edgewise arch or a universal assemblage of Atkinson, in conjunction with intermaxillary elastics during the day, and the occipital, pitted against the maxillary first molars, at night. This relieves the strain on the mandibular resistance units and the transseptal fibers between the first and second maxillary molars, where the second maxillary molars move much more quickly than the first.

In cases where the maxillary buccal segments have drifted forward, blocking out canines, either unilaterally or bilaterally, the method just described is employed with equal success.

In mixed dentitions, where the anchorage units are weak because of the lack of approximal contact of the teeth, the use of intermaxillary elastics would not be practicable, whereas the occipital and cervical anchorage would fill every requirement.

The distal movement of maxillary incisors may be accomplished with this type of headgear arch by soldering four long spurs to the incisal segment and bending them lingually over the incisal edges of the central and lateral incisors to prevent the arch wire from creeping gingivally and elongating these teeth. Distal pressure on the labioincisal angle of these teeth, when the elastics are applied, tips the crowns lingually without displacing the root apices labially.

The distal movement of mandibular molars may be accomplished with this type of headgear apparatus with a good deal of success. In fact, its use in the mandibular arch is almost as versatile as in the maxillary.

In comparing methods of application with Dr. Richard Lowy, of Chatam, New Jersey, the essayist was informed that he, also, had been highly successful with the distal movement of mandibular molars in the treatment of Angle Class III malocclusion.

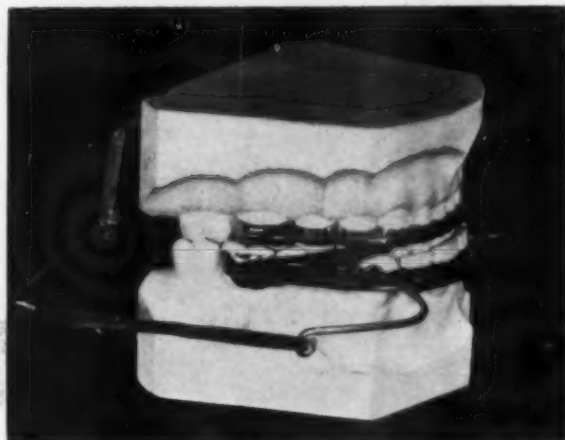


Fig. 5.—(From Dr. Waldron.)

In a bimaxillary protrusion, where the distal movement of both maxillary and mandibular teeth is desired, the occipital cervical anchorage may be employed. Here, the winged-type arch is used and stopped at the maxillary molars. A 0.040 arch is conformed to the labial surfaces of the mandibular teeth, to which are soldered two intermaxillary hooks, distal to the canines, and stops in front of the molar tubes. Intermaxillary light Class III elastics are employed, in conjunction with the extraoral elastics, which exert from three to four times as much force as the intraoral. It is therefore obvious that the extraoral influence is sufficient to move the maxillary teeth distal, which are harnessed to the mandibular teeth, carrying them distal also. (Fig. 5.)

Photographs of the casts and the patient, treated by Dr. Oppenheim for less than three years with occipital and cervical anchorage, along with a bite plate having no inclined plane, speaks for the possibilities of this form of strategy (Figs. 6, 7, and 8). Due to the traveling distance from the home of the patient to the doctor's office, Dr. Oppenheim saw this patient only twice a year, totalling approximately six or seven visits in all.

In a recent letter from Dr. Atkinson, he mentioned that in every case where he had used occipital anchorage the results were most gratifying, but he advised the use of light rubbers.



Fig. 6.—Patient before and after treatment. (From Oppenheim.)

Fig. 7.

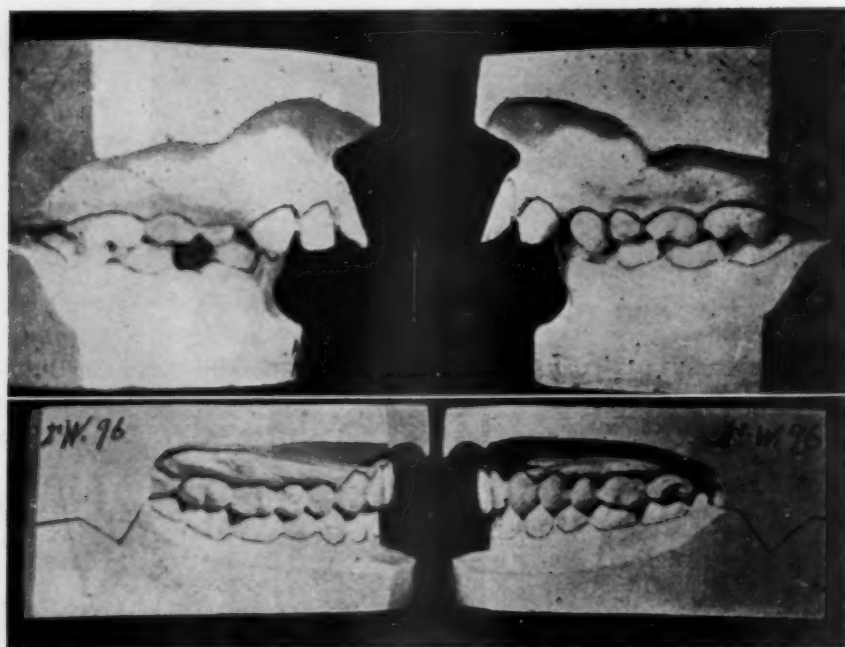


Fig. 8.

Fig. 7.—Casts before treatment. (From Oppenheim.)

Fig. 8.—Casts twenty-eight years after treatment. (From Oppenheim.)

Dr. Ernest L. Johnson, of San Francisco, California, describes an apparatus for the distal movement of maxillary and mandibular teeth, simultaneously, with occipital and cervical anchorage, in the November, 1943, issue of the *AMERICAN*

JOURNAL OF ORTHODONTICS AND ORAL SURGERY. In this article he also describes a method for the mesial movement of posterior teeth.

The use of occipital and cervical anchorage in conjunction with traction upon the chin was first introduced by Dr. Charles S. Tomes, and reported in the *British Journal of Dental Science*, May, 1873, as follows:

"As soon as the upper teeth had been drawn outward so that the arch was wide enough to allow of their passing outside the lower teeth, pressure was brought to bear in order to close the front part of the mouth by means of a simple arrangement of elastic bands. A circular air-cushion was adapted to the chin and connected by strong pieces of elastic with a cloth band passing over the top of the head; the whole was kept in place by two pairs of ribbons which were tied at the back of the head. At first some little trouble was experienced, owing to the skin of the chin becoming tender under the heavy pressure, but this was combatted by the use of spirit lotions, and by putting slightly oiled lint between the air-pad and the skin. The apparatus was worn constantly at night, and also during a considerable part of the day, the teeth being kept from falling back into their former positions by a light retaining plate. At the time when this apparatus was first adjusted, the only teeth which came into contact were the upper and lower second molars and second bicuspsids.

"For a few weeks no very marked effect was produced, save slight pain in the region of the temporo-maxillary articulation; but after that, the gap between the upper and lower incisors diminished each week by an amount that could be measured, and at the expiration of six months from the commencement of treatment (the elastic bandage having been worn for about four months), not only had the gap entirely closed, but the upper central incisors had been made to over-lap to the extent of one-sixteenth of an inch, while the laterals and the canines also over-lap and antagonize. Not only has this patient's appearance been very greatly improved by the closure of the mouth, but the power of mastication, which before treatment was necessarily very imperfect, has been most materially improved."



Fig. 9.—(Diagram from Oppenheim.)

The above led to the use of the chin cap described by Calvin Case and now used, by those who employ extraoral anchorage, in a refined and convenient form. This chin cap is constructed of lightweight metal, highly polished and chrome plated. The periphery is perforated for the reception of special pins which act as hooks for the attachment of rubbers. These chin caps are made in three sizes: small, medium, and large. When padded on the inside with gauze or absorbent cotton, they fit almost any chin.

The chin cap, as an auxiliary with the occipital, has many practical uses in our field of endeavor.

Following is a communication from Dr. Oppenheim, dated April 15, 1943.

"In Class III cases, where the chin cap, in order to exert a distal pressure, is not advisable on account of the advanced age of the child (six years or more) the upper denture can easily be brought forward to counterbalance the protruding chin. This method has no age limit. I treated in this way successfully patients forty years old. All you have to do: An upper lingual arch, well fitting in the front at the gingival margin; the two first molars have hooks at the mesiobuccal corners. [Fig. 9.]

"The chin cap is fastened by light elastics (b) or even only by non-elastic cords to the head cap. From the chin cap go two rods upward (r) corresponding in distance to the angles of the mouth and having several spurs (s) at the upper end between which the elastics from the molars are hooked in; this prevents a sliding of the elastics and a cutting into the lips.

When the elastics are in place they tend to press the rods against the lips; this is counteracted by a non-elastic ribbon (a) that is fastened in the neck so tight only, that the pressure on the lip is eliminated. In grown up people this device is supported by intermaxillary *very light* elastics (stamped out of rubber dam) during the day; *once or twice* a week; for this purpose a lower labial *heavy* arch with intermaxillary hooks is adjusted and is removed in the evening.

"In front of the tube is soldered a spur to prevent the arch from pressing against the front teeth. These very light elastics will not tilt the lower molars.

"Only *real* open bite cases can be successfully treated by the head cap, that is cases where you have occlusal contact on the last molars. If the open bite starts in the region of the bicuspid it will not work; for the lever to bend the angle of the mandible must be quite long. The cases starting in the bicuspid region are caused by some habit too, which you mostly cannot overcome and the failure is unavoidable. In real open bite cases the obtuse angle has to be changed to a more right angle."



Fig. 10.—(From Atkinson.)

Fig. 11.

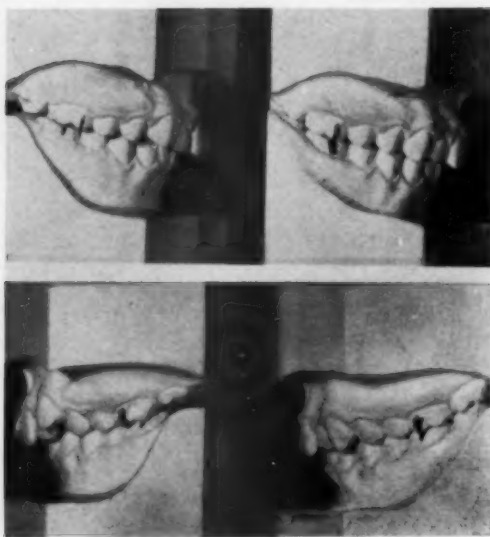


Fig. 12.

Figs. 11 and 12.—Casts showing distal movement of maxillary buccal segments. Maxillary canines in good position.

Attention is directed to the change in the thickness of the cortical plate at the notch located on the inferior border of the mandible, making the bend possible (Fig. 10).

The writer wishes to quote from an excellent paper on anchorage in the treatment of malocclusion, which was read by Dr. F. Copeland Sheldon, of Kansas City, Missouri, before the annual midyear meeting of the Chicago Dental Society and published in the *Journal of the American Dental Association*, February, 1944. "The use of extraoral anchorage is nothing new to the science of orthodontics. It has been used and discarded many times during the course of progress of our profession. Its use as a means of moving teeth has not been given a thorough test in my practice, although it might be said to offer unlimited possibilities. As a means of augmenting anchorage, however, it is one of our most valuable aids."

It is most regrettable that so many orthodontists are aware of the possibilities of occipital and cervical anchorage, but have not given this mechanism a thorough test in their practice. Should they do so, they would be amply rewarded for their efforts in the results achieved.

Figs. 11 and 12 are the casts of a girl, aged 12 years, presenting a forward drifting of the right and left maxillary buccal segments, blocking out the left maxillary canine, and an overlapping of the right central incisor over the left. The maxillary right and left buccal segments were moved distally and the normal mesiodistal relationship restored. This was accomplished with extraoral force and occipital anchorage applied only at night. There were no appliances worn during the day.

In conclusion, it is with a great deal of satisfaction that the author observes that this type of anchorage, which has been in disuse for a long time, is once again coming to the fore. Its appearance on the scientific programs of our meetings and the consequent publication in the journals should be encouraged so that its application may be developed to a wider use.

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THE MAXILLATOR: A NEW INSTRUMENT FOR MEASURING THE
FRANKFORT-MANDIBULAR BASE ANGLE, THE INCISOR-
MANDIBULAR BASE ANGLE, AND OTHER COMPONENT
PARTS OF THE FACE AND JAWS

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INTRODUCTION

THE Maxillator (maxilla-meter) provides a new means for measuring the face and its components, including the jaws, jawbones and their parts, processes, and anthropologic, orthodontic, and other bone and soft tissue landmarks. By using this instrument, various face and jaw measurements, including angles and lines, can be taken directly on the face of the living subject or patient as well as on wet or dry specimens and read directly on the millimeter scales and protractors which form integral parts of the instrument itself.

The use of the Maxillator does not entail the employment of casts of the face and jaws, x-ray films, photographs, facial tracings or drawings, head-positioning machines, x-ray machines, photographic equipment, and other time-consuming methods and costly equipment now employed in making facial measurements in orthodontics, prosthetics, and in other fields such as physical anthropology, comparative dental anatomy, statistical studies of growth and development and other changes of the face and jaws.

This instrument can be used in the practice of orthodontics for the purpose of obtaining measurements on the growth and development of the face before beginning treatment, and as an aid in diagnosis and classification in outlining and planning treatment; to check on the progress of treatment and in determining prognosis.

The Frankfort-mandibular base angle, the importance of which in orthodontics was first described by Tweed, who also established definite criteria for the use of this angle in orthodontic diagnosis and treatment planning, can be obtained accurately and quickly with the Maxillator by taking the measurements directly on the patient. The incisor-mandibular plane angle, first described and employed in orthodontic diagnosis by Margolis to measure the variation in the 90-degree relationship of the mandibular incisors to the mandibular base plane, also can be obtained directly on the patient by means of the incisor-mandibular plane Angulator, which is part of the Maxillator.

By using the Maxillator it is possible to obtain other facial measurements and indexes to serve as guides in restoring facial contours by means of plastic operations, as in skin and muscle grafting, and in the construction of full or partial, fixed or removable dentures and dental restorations.

This instrument can be employed also for obtaining data useful in identification of faces, skulls, and their component parts.

DESCRIPTION OF THE MAXILLATOR

The Maxillator Body Piece (Fig. 1).—This is a right-angle triangle with each of the two sides 6" long. The triangle can be made of any rigid or semirigid material of any thickness. I have found the use of a plastic material such as plexiglass $\frac{3}{16}$ " in thickness to be most suitable.

A $\frac{3}{16}$ " hole is made through the thickness of the triangle where two lines projected at a distance of $\frac{3}{8}$ " on each side of the 90-degree angle, and parallel with the sides, cross each other.

A slot $\frac{3}{16}$ " wide and 4" long is made through the thickness of the triangle along each of the lines by which the hole was located. The slots run parallel with the 6" sides and terminate at 1" from each end of the 6" sides of the triangle.

A millimeter ruler is inscribed on each of the 6" sides of the triangle, beginning with the diameter of the $\frac{3}{16}$ " hole and running to approximately 12 cm. along each of the 6" sides of the triangle.

The Mandibular Base Piece (Fig. 2).—This is made of the same material or materials as Fig. 1. Fig. 2 consists of three attached parts:

Part 1. The vertical protractor piece.

Part 2. The horizontal fin.

Part 3. The incisor-mandibular plane angulator.

Part 1 of Fig. 2 is 6" long. On the right side is a circle $2\frac{1}{2}$ " in diameter with a $\frac{3}{16}$ " hole running through the thickness of the material at the center of the circle. The top of this part then extends a distance of $2\frac{1}{2}$ " to the left of the circumference of the circle on the same horizontal plane as the middle of the hole at the center of the circle. The top of Part 1 of Fig. 2 is then extended upward $\frac{1}{2}$ " at right angles to the $2\frac{1}{2}$ " extension and is continued to the left for 1" at right angles to the $\frac{1}{2}$ " extension and on to the left edge of the part.

On the same horizontal line which runs through the center of the hole in the center of the $2\frac{1}{2}$ " circle on the right side of Part 1 of Fig. 2 and identical with the $2\frac{1}{2}$ " line running to the left of the circumference of the circle, a $\frac{3}{16}$ " hole is made through the thickness of the material, with its center at a distance of $\frac{1}{2}$ " from the left end side of Part 1 of Fig. 2. From the center of this hole an arc $1\frac{1}{4}$ " in diameter is described running from the left edge of the part to $\frac{3}{4}$ " below the $2\frac{1}{2}$ " horizontal extension on top which runs left to the circumference of the $2\frac{1}{2}$ " circle on the right. The bottom of Part 1 of Fig. 2 is then completed by a line running from the arc on the left to the circle on the right at a distance of $\frac{3}{4}$ " below the $2\frac{1}{2}$ " extension at the top.

A protractor is inscribed on the circumference of the arc on the left side with 0° at the horizontal line which passes through the center from which the arc was described and the center of the hole in the middle of the circle on the right side. The protractor is divided into degrees running downward to the edge on the left-end side of Part 1 of Fig. 2.

Fig. 1 is then placed in such a position that one of the 6" sides is horizontal and the other 6" side extends downward vertically to the right. Part 1 of Fig. 2 is then placed under Fig. 1 (as shown in Fig. 4) so that the hole in the center of the circle on the right side of Part 1 of Fig. 2 is directly under the 4" slot which runs parallel with the vertical 6" side of Fig. 1. While the parts are held in this relationship, Part 1 of Fig. 2 is rotated so that a line running through the center of the circle on the right side and the center of the hole from which the arc on the left side of Part 1, Fig. 2 was described will be parallel with the top 6" side of Fig. 1. This makes the fin or Part 2 of Fig. 2 parallel with the horizontal 6" side of Fig. 1 (also shown in Fig. 4). A mark is now made on the bottom of the circumference of the circle on the right side of Part 1, Fig. 2, where the edge of the vertical 6" side of Fig. 1 falls on it.

Fig. 1 is removed from Part 1 of Fig. 2, and a protractor is inscribed on the circumference of the $2\frac{1}{2}$ " circle on Part 1 of Fig. 2, with 0° at the mark which indicates where the edge of the vertical 6" side of Fig. 1 crossed the lower circumference of the circle on the right side of Part 1 of Fig. 2. The protractor is graduated in degrees, runs to the right, and stops at the horizontal line which passes through the diameter of the circle.

A second protractor is inscribed on the top of the circumference of the circle on the right-end side of Part 1 of Fig. 2. The 0° begins at the point on the top circumference which is on a line running at right angles to the fin or Part 2 of Fig. 2 and through the vertical diameter of the $2\frac{1}{2}$ " circle as well as through the vertical center of the 4" slot which runs parallel with the vertical 6" side of Fig. 1. This protractor is graduated in degrees and runs right and left to the horizontal line which passes through the diameter of the $2\frac{1}{2}$ " circle on the right side of Part 1 of Fig. 2.

Part 2 of Fig. 2, the fin, can be made of the same material or materials as Fig. 1. Part 2 of Fig. 2 is $4\frac{1}{2}$ " long. One end of this part is cut $\frac{3}{4}$ " in width at right angles to the $4\frac{1}{2}$ " side, and the other end is $2\frac{1}{2}$ " in width also cut at right angles to the $4\frac{1}{2}$ " side.

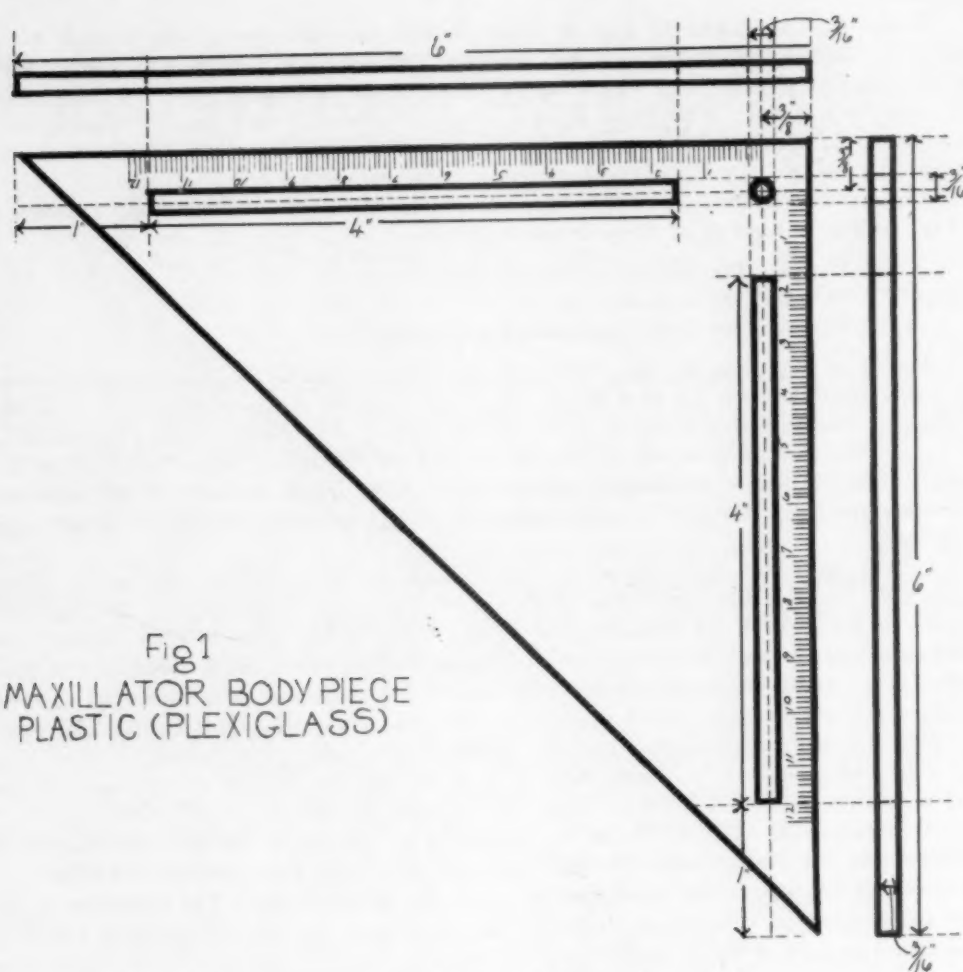


Fig 1
MAXILLATOR BODY PIECE
PLASTIC (PLEXIGLASS)

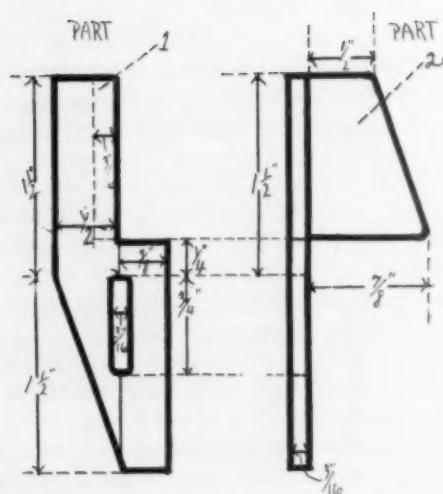
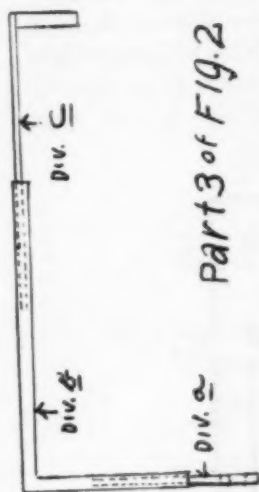


Fig 3
RAMUS PIECE



Part 3 of Fig. 2

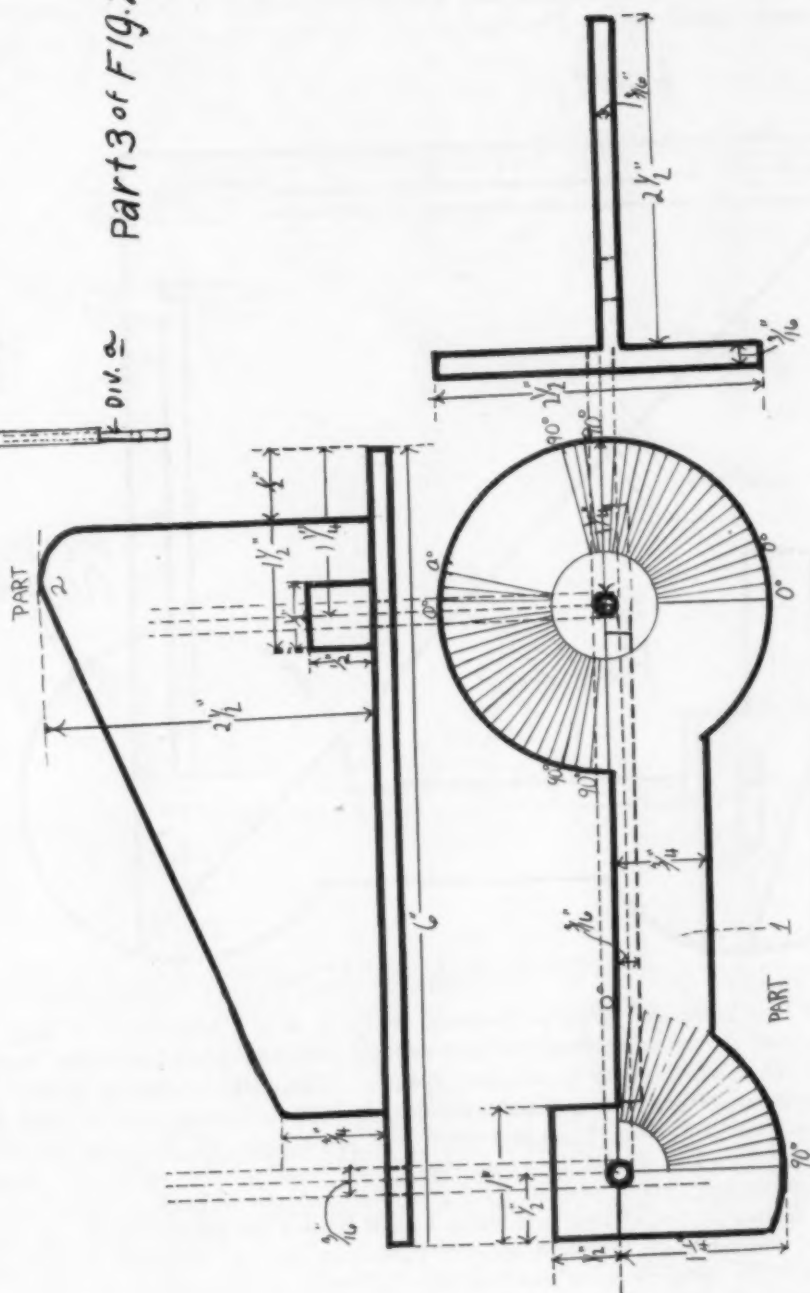


Fig 2 BASE PIECE

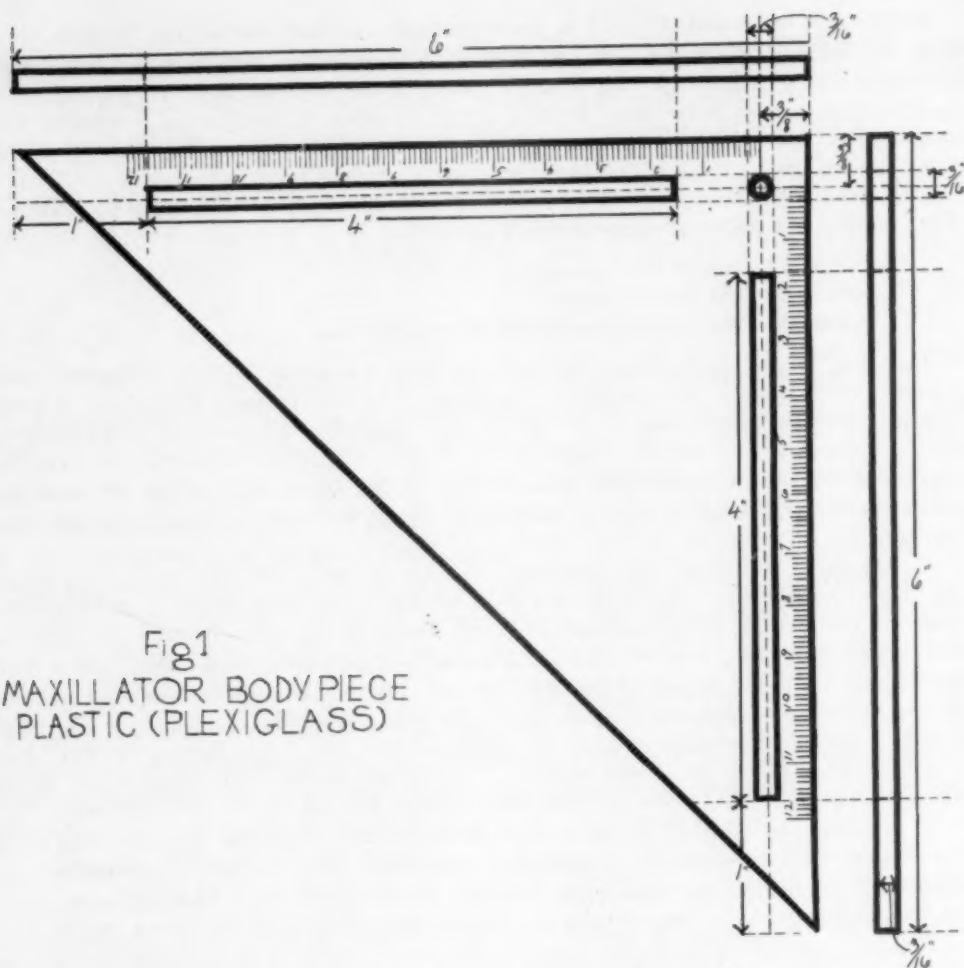


Fig. 1
MAXILLATOR BODY PIECE
PLASTIC (PLEXIGLASS)

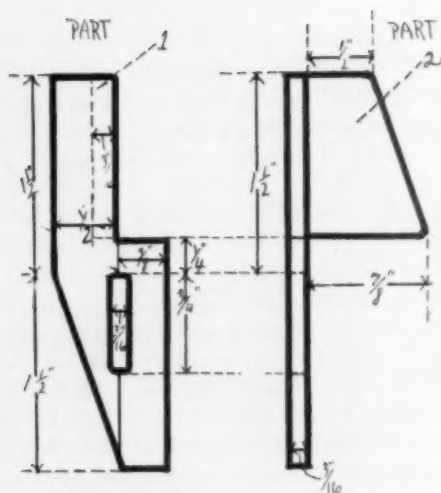


Fig. 3
RAMUS PIECE

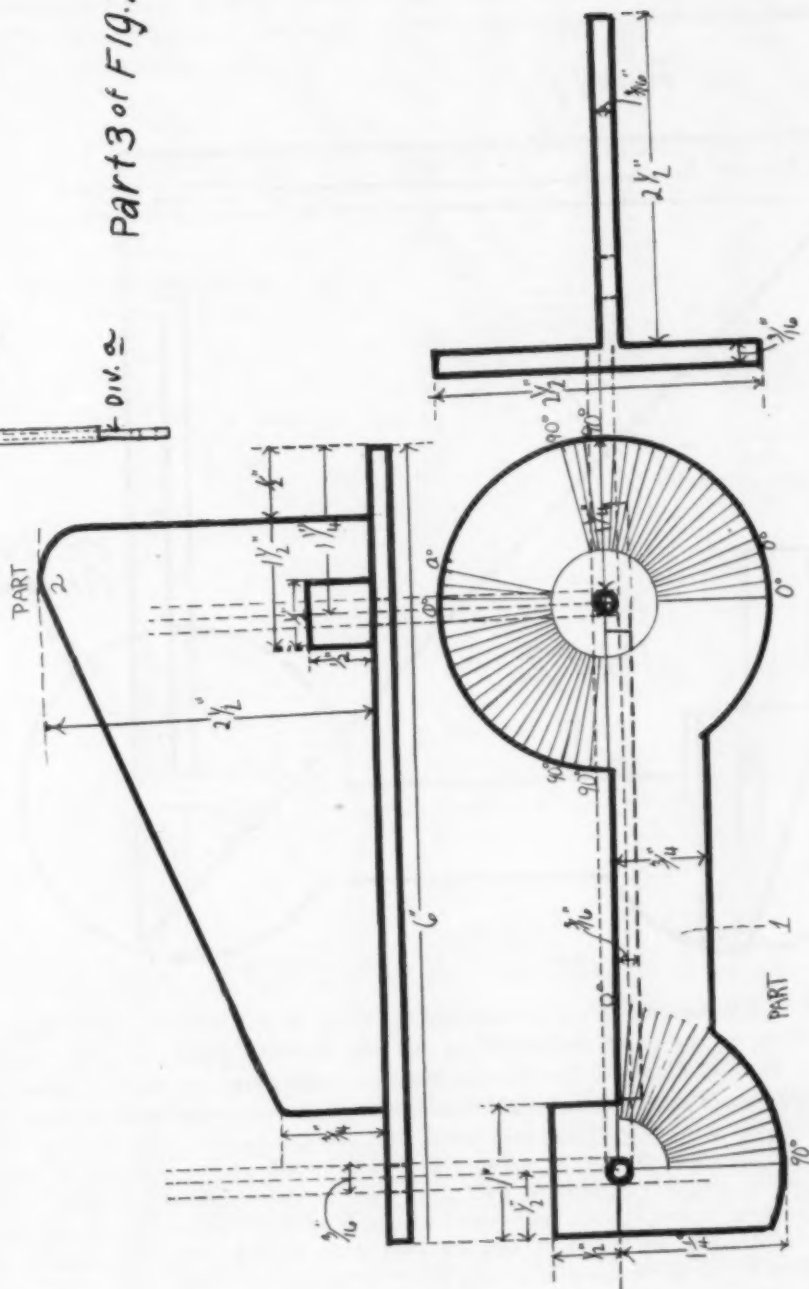
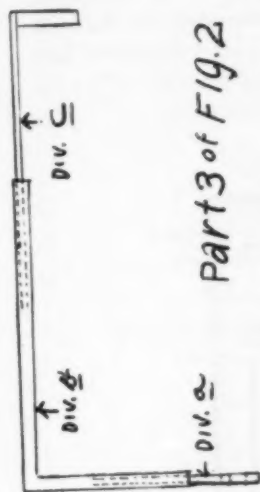
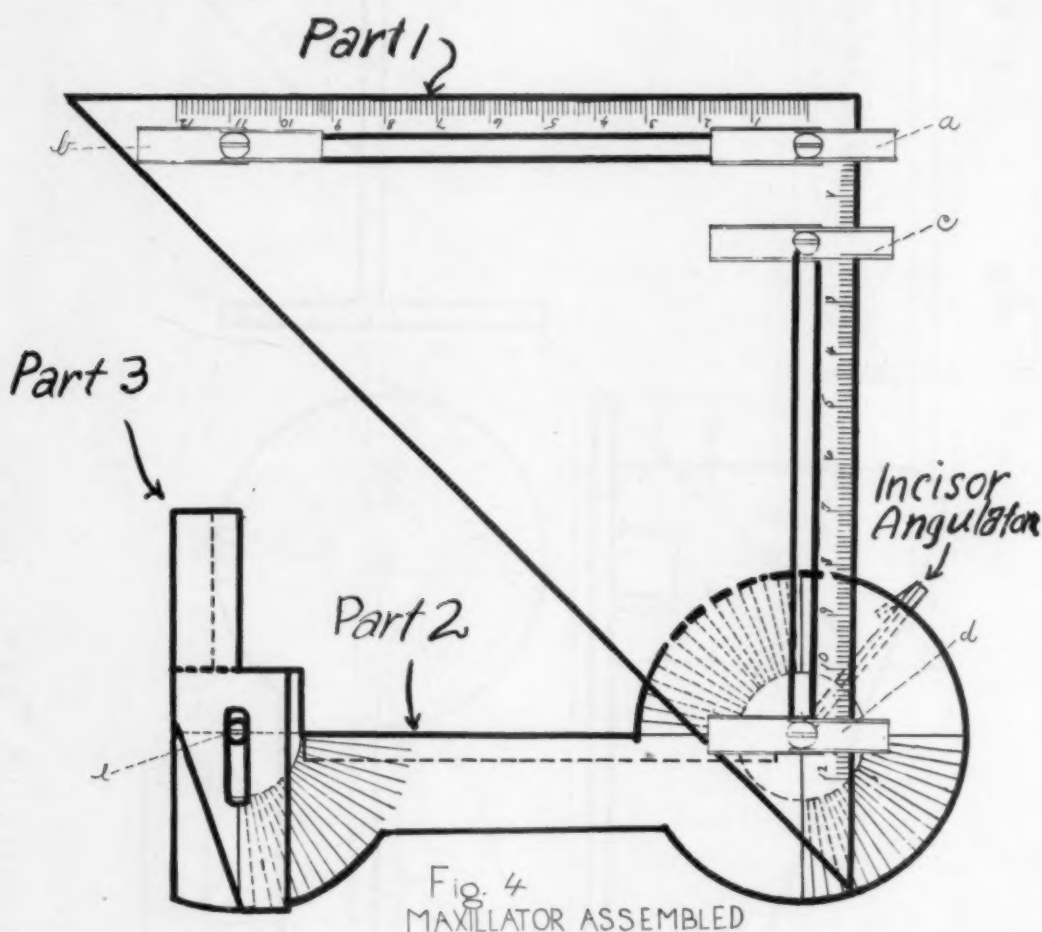


Fig 2 BASE PIECE

The ends are now connected by a straight line which goes from the $2\frac{1}{2}$ " side to the $\frac{3}{4}$ " side and makes up the fourth side of this part. The angles between the ends and the tapering side are leveled to $\frac{1}{2}$ " on the $2\frac{1}{2}$ " side and $\frac{1}{8}$ " on the $\frac{3}{4}$ " side.

Part 2 of Fig. 2 is attached to Part 1 of Fig. 2 with the $4\frac{1}{2}$ " side right below a line running through the centers of the $2\frac{1}{2}$ " circle on the right and the arc on the left, beginning at a point $\frac{1}{2}$ " from the horizontal circumference of the circle on the right side of Part 1 of Fig. 2. Part 2 of Fig. 2 is attached to Part 1 of Fig. 2 on the side away from the observer (see Fig. 2). The $2\frac{1}{2}$ " end of the fin is toward the circle and the $\frac{3}{4}$ " end of the fin is toward the arc on Part 1 of Fig. 2. A square $\frac{1}{2}$ " by $\frac{1}{2}$ " is cut from Part 2 of Fig. 2 on the $4\frac{1}{2}$ " edge so that the cut-out square is equidistant from the sides of the hole in the center of the circle on the right-hand side of Part 1, Fig. 2. This will permit adjustment of screw assembly *d* (see Fig. 4).



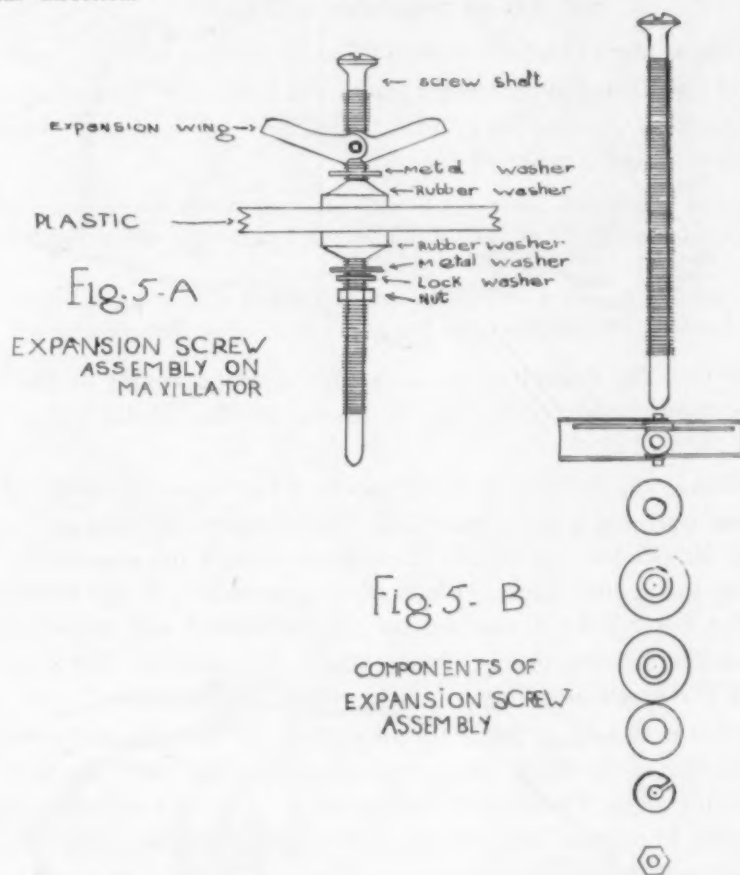
Part 3 of Fig. 2 consists of three divisions. Division *a* is a half-round, 1" long, 15 gauge post soldered to the outer circumference and on the same plane of a flat washer with inner hole $\frac{3}{16}$ " in diameter to fit over the $\frac{1}{8}$ " wide threaded rod of screw assembly *d* (see Fig. 4). Division *b* is a right angle made of a half-round tube with the flat surface to the outside. One arm of the half-round tube triangle is $1\frac{1}{2}$ " long and the other arm is $2\frac{1}{4}$ " long. The $\frac{3}{16}$ " washer to which the 1" half-round wire is soldered is placed over the rod of screw assembly *d*, next to Part 2 of Fig. 2 or the vertical protractor piece, so that the flat surface of the 1" long half-round wire is toward the protractor on the upper circumference of the circle on the right side of Part 1 of Fig. 2. The lock washer and nut are then added to complete screw assembly *d*.

A line is grooved on the vertical center of the flat surface of the short arm of the triangle or division *b* of Part 3 of Fig. 2. The short arm of the triangle is now placed over the 1" half-round wire. Division *c* of Part 3 of Fig. 2 is a half-round wire 2" long which fits into the horizontal part of Division *b*. At the free end of Division *c* a $\frac{1}{2}$ " piece of half-round wire is soldered at right angles so that at its free end it is $\frac{1}{16}$ " off center from

the vertical $1\frac{1}{4}$ " arm of division *b* (see Part 3 of Fig. 2). The flat surface of the $\frac{1}{2}$ " extension is toward the observer when the right angle of division *b* is toward the left hand.

The Ramus Piece (Fig. 3).—This is made of the same material or materials as Fig. 1. Fig. 3 consists of two attached parts. Part 1 of Fig. 3 is 3" in length. Part 2 of Fig. 3 is $1\frac{1}{4}$ " long and is attached at right angles to Part 1 of Fig. 3.

Part 1 of the ramus piece is cut $\frac{1}{2}$ " wide on top. The right side is cut at right angles to the top and extends for $1\frac{1}{4}$ ", then to the right at right angles $\frac{3}{8}$ ", then downward $1\frac{1}{4}$ " to the bottom which runs to the left $\frac{3}{8}$ " at right angles to the right side. The top and bottom of the piece on the left side are joined by a line running at right angles from the top downward for a distance of $1\frac{1}{2}$ " then connecting with the bottom at an angle. A $\frac{3}{16}$ " wide by $\frac{3}{4}$ "-long slot is cut in this piece at a distance of $1\frac{1}{2}$ " from the top and $\frac{1}{2}$ " from the vertical center of the slot to where the left-end side would be if that had been continued in a vertical direction.



Part 2 of Fig. 3 is $1\frac{1}{4}$ " long vertically. The top is $\frac{1}{2}$ " wide and at right angles to the $1\frac{1}{4}$ " side. The bottom is $\frac{7}{8}$ " wide and at right angles to the $1\frac{1}{4}$ " side. The fourth side of Part 2 of Fig. 3 connects the top and bottom sides.

Part 2 of Fig. 3 is attached at the right side and at right angles to Part 1 of Fig. 3 and extends from the top of the piece downward and away from the observer.

The Maxillator Assembled (Fig. 4).—The screw rod assembly *a* (see also Figs. 5A and 5B) is placed through the hole at the 90-degree angle at the top right side. Screw assembly *b* is placed through the top horizontal slot, and screw assembly *c* is placed through the vertical slot.

Part 1 of Fig. 2 is placed under Fig. 1 so that the threaded rod of screw assembly *d* with toggle, metal and rubber washers can be adjusted through the vertical slot on the right side of Fig. 1 and through the hole in the center of the circle on the right side of Part 1 of Fig. 2, extending into the cut-out square of the fin, or Part 2 of Fig. 2, and through the washer to which the 1" half-round wire, or division *a* of Part 3 of Fig. 2, was soldered. The lock washer and nut are now added to complete screw assembly *d*.

Part 1 of Fig. 3 is placed so that the screw assembly *e* can be adjusted through the slot of Part 1 of Fig. 3 and through the hole at the center of the arc on the left side of Part 1 of Fig. 2.

Expansion Screw Assembly on Maxillator (Fig. 5A).—This shows the screw rod $\frac{1}{8}$ " wide and 3" long with mushroom head and tapered end. The expansion wing (toggle, Star No. 3006- $\frac{1}{8}$ snap in) is placed on the screw rod with the wings to the head of the screw. A metal washer is placed over the screw rod. A rubber or fabric washer is placed over the screw rod, with the flat surface to the end of the screw rod. The screw rod is inserted into the Maxillator. A rubber washer is then placed with the flat side away from the end. This is followed by a thin metal washer, a lock washer, and a nut.

The components of the screw assembly in order of placement on the Maxillator are shown in Fig. 5B.

METHOD OF USING THE MAXILLATOR

1. *To Obtain the Frankfort Plane.*—The Frankfort plane is used to orient the head or the skull in the horizontal plane. A horizontal line which runs from the orbital point to the tracion point is the line along which the head must be orientated when in the Frankfort plane.

a. *To obtain the orbital point*, mark with skin pencil the lowest point on the lower border of the orbit, directly below the pupil when the eye is open and the patient is looking straight ahead.

b. *To obtain the tracion point*, mark with skin pencil a point on the notch just above the tragus of the ear. The tracion point lies 1 to 2 mm. below the spina helix.

2. *To Obtain the Orbital Plane.*—A line at right angles to the Frankfort plane at the orbital point (orbitale) is known as the orbital plane (Figs. 6A and 6B).

3. *To Obtain the Length of the Frankfort Plane on a Straight Line.*—Adjust the screw assembly *a* to extend all the way through the hole at the 90-degree angle on the Maxillator. Hold the Maxillator so that the end of the screw assembly *a* is at the orbital point. Place screw assembly *b* at the tracion point as shown in Fig. 6B. Take a reading on the horizontal millimeter scale of the Maxillator at the tracion point, which should pass through the point of screw assembly *b* while screw assembly *a* is held at the orbital point.

4. *To Obtain Length of the Face on a Straight Line From Orbital Point to the Lower Border of the Mandible.*—Ask the patient to clench the teeth together. Place Maxillator along Frankfort plane as in 3. Take a reading on the vertical millimeter scale at a point opposite the lower border of the mandible.

5. *To Obtain Mesiodistal Relation of the Maxillary Dental Arch to the Orbital Plane.*—Place the Maxillator with screw assembly *a* at the orbital point and screw assembly *b* at the tracion point as in 3. Adjust screw assembly *c* to the incisal edge of the maxillary tooth that lies opposite the point of this screw assembly.

6. *To Obtain the Relationship of the Mandibular Teeth to the Orbital Plane.*—Place the Maxillator as in 5. Adjust the screw assembly *c* to the mandibular tooth that lies opposite the point of this screw assembly.

7. *To Obtain the Frankfort-Mandibular Base Angle.*—

The patient is seated upright with the head on a line with the torso. The fin (Part 2 of Fig. 2) is placed well under the mandible and is pressed gently but firmly against the lower border of the mandible by pushing upward with the hand at the center of the under-surface of the fin. The fin thus becomes the mandibular base plane, i.e., the plane tangent to the most dependent points of the lower border of the mandible. The Frankfort-mandibular base angle is the angle formed by the Frankfort plane and the mandibular base plane.

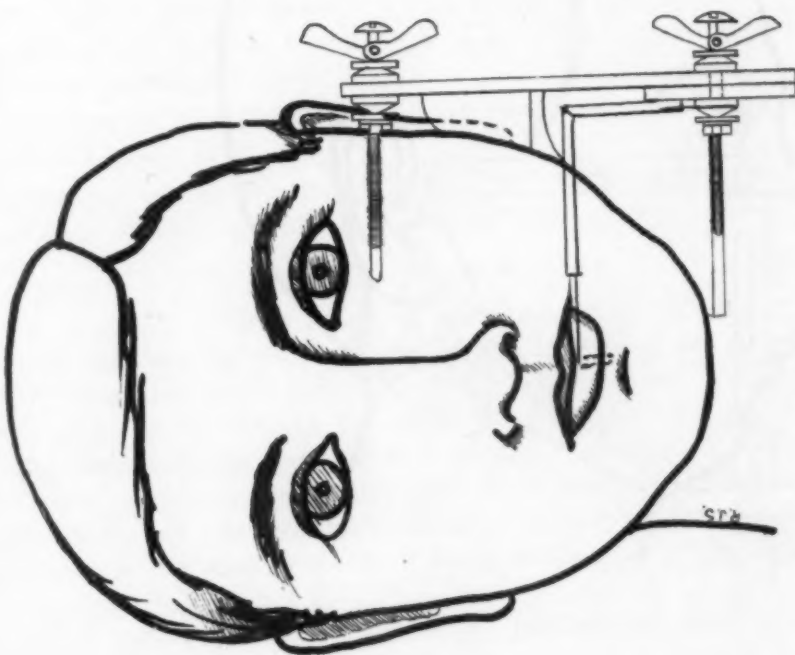


Fig. 6-A
MAXILLATOR ADJUSTED TO
FACE. FRONT VIEW

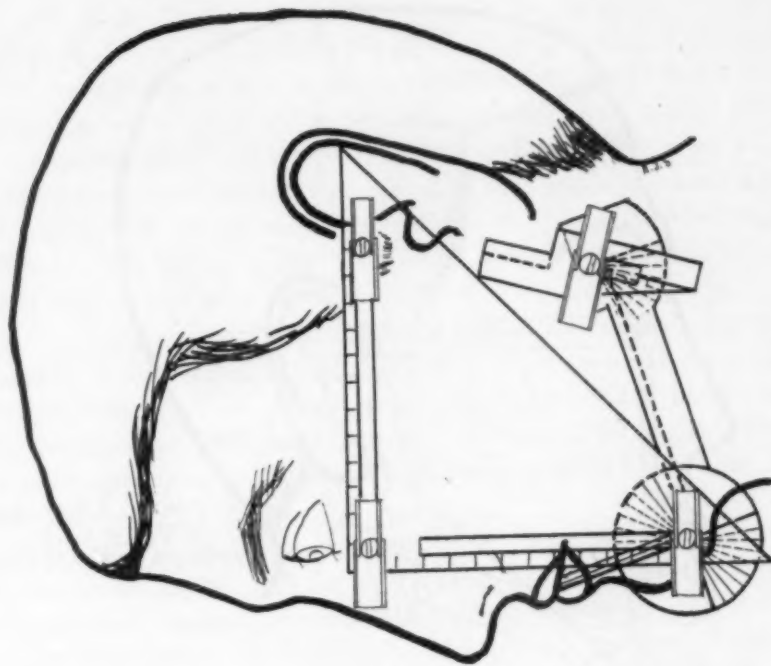


Fig. 6-B
MAXILLATOR ADJUSTED
TO FACE. SIDE VIEW

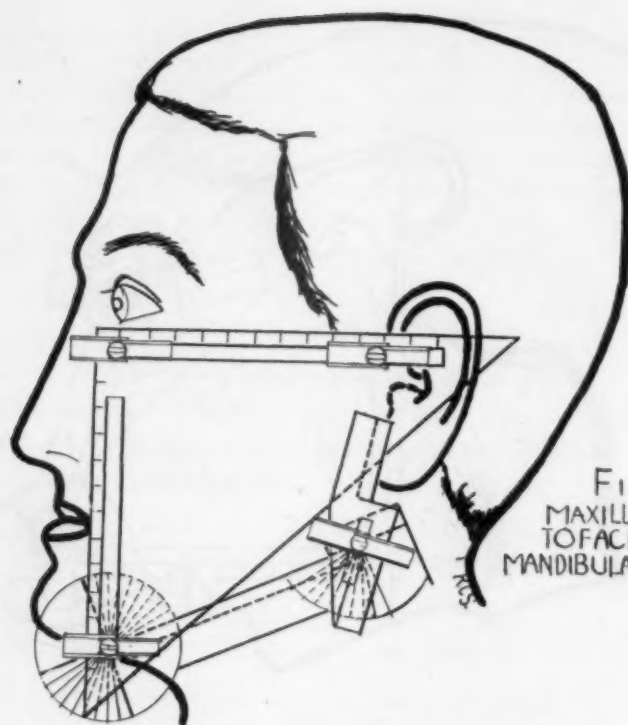


Fig. 7
MAXILLATOR ADJUSTED
TO FACE WITH
MANDIBULAR PROTRACTION

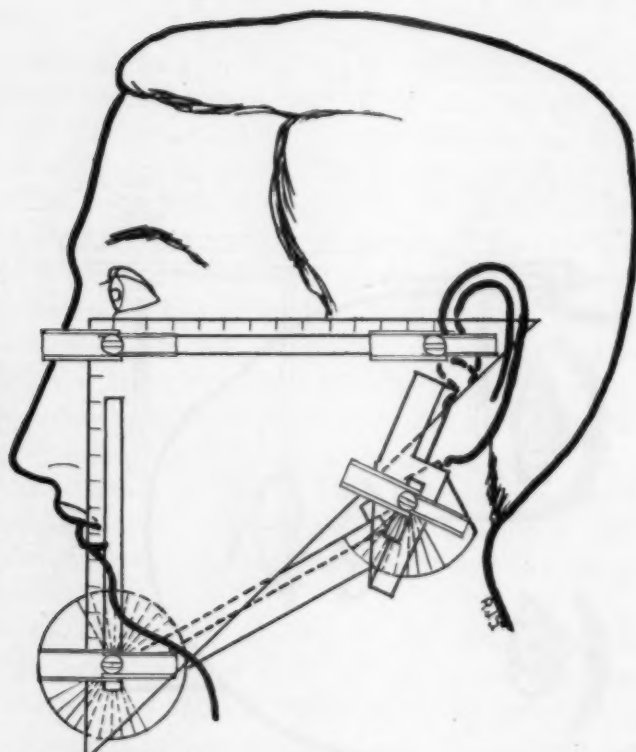


Fig. 8
MAXILLATOR ADJUSTED TO
FACE WITH MAXILLARY PROTRACTION

Place the Maxillator with screw assemblies *a* and *b* at the orbital and trigion points, respectively, as explained in 3. Sight along vertical millimeter scale for the length of the face at the orbital plane as explained in 4. Adjust screw assembly *d* to this point. Adjust the fin of Part 2 of Fig. 2, the mandibular base piece, so that it lies at and is pressed against the lower border of the mandible, while screw assemblies *a* and *b* are maintained at their respective points on the face indicating the Frankfort plane (Figs. 6A and 6B).

The size of the Frankfort-mandibular base angle is shown on the protractor at the bottom of the circumference of the 2½" circle on the right side of the mandibular base piece, or Part 1 of Fig. 2, where it is crossed by the edge of the vertical 6" side of Fig. 1, or the maxillator body piece (see Fig. 2 and Fig. 6B).

8. *To Obtain the Gonion Angle.*—Place the ramus piece in back of the ramus and the mandibular base piece against the lower border of the mandible as explained in 7. Take a reading on the protractor on the arc on the left side of the mandibular base piece where it is crossed by a line extended through the center of the vertical dimension of the slot in Part 1 of Fig. 3 (see Figs. 2, 3, and 6B).

9. *To Obtain the Incisor-Mandibular Plane Angle.*—Adjust the vertical extension on Division *c* of Part 3 of Fig. 2 to lie against the lingual surface of the mandibular central incisor, right or left, whichever one is more typical of the inclination of the other mandibular incisors. Adjust Division *b* of Part 3 of Fig. 2 over the horizontal end of Division *c*. Mark a point on the face directly under the vertical part of Division *b* of Part 3 of Fig. 2. Adjust Division *a* of Part 3 of Fig. 2 so that it fits into the tube of the vertical part of Division *c* of Part 3, Fig. 2. While maintaining the vertical part of Division *b* of Part 3 of Fig. 2 over the mark made under it on the side of the face, adjust the fin, or Part 2 of Fig. 2, to lie against the base of the mandible as indicated for 7. Take a reading on the top protractor where the vertical midline of the vertical part of Division *b* of Part 3 of Fig. 2 crosses the protractor. This is the incisor-mandibular plane angle.

The use of the Maxillator on the face with mandibular protraction is shown in Fig. 7.

The use of the Maxillator on the face with maxillary protraction and mandibular retraction is shown in Fig. 8.

SUMMARY

A new instrument is presented by means of which various face and jaw measurements can be obtained directly on the subject or patient without the use of time-consuming methods and costly apparatus. Among the measurements that can be obtained by using this instrument are the Frankfort-mandibular base angle, the importance of which in orthodontics had been described by Tweed; the incisor-mandibular plane angle described by Margolis; and other angles, lines, and relationships of value in orthodontics, prosthetics, and fields which require the measurements of facial landmarks, lines, and angles.

AN ADJUSTABLE LOCKING BRACKET FOR USE WITH MULTIPLE BAND THERAPY

J. E. LASKIN, D.D.S., CLEVELAND, OHIO

IN MULTIPLE band therapy for the correction of malocclusion, the engagement of the arch wire to the brackets is obtained by ligating or by lock pins. In rectangular arch wire therapy, the use of second order bends or tip-back bends in conjunction with local torque bends or progressive mass torque bends is employed.

It has been conceded that an arch wire, embodied with a combination of tip-back bends and progressive torque bends, is so contorted that it is impossible to be sure that the results desired will be obtained.

The technique for bending an arch wire with a combination of tip-back bends and torque bends is both complex and confusing and tedious.

Ligating and forming tip-back bends are two components of the edgewise arch technique which, all who employ them will agree, are tedious, time consuming, and, because of the complexities involved and the chair time they consume, definite factors in the number of cases that can be treated annually.

In this paper, an adjustable locking bracket is presented for the first time: a bracket which will eliminate almost completely the two time-consuming factors mentioned and will find, it is believed, other practical applications in the edgewise arch technique, where it shows promise of projecting a more simple and precise technique. It may be used also in other labial arch techniques where bands are placed on the teeth. (Figs. 1 and 2.)

The Adjustable Locking Bracket will eliminate the need for gingivo-occlusal bends in the arch wire, such as the mesial and distal second order bends for mass movements of the buccal segments. It will eliminate gingivo-occlusal bends now made in arch wires to produce root and crown movements of the incisors, and it will almost entirely eliminate the use of ligatures, because it locks the arch wire in the bracket channel when properly adjusted.

The Adjustable Locking Bracket is a one-cylinder machine, utilized to force power into the arch wire for the movement of a tooth or teeth (Fig. 3).

By simply adjusting the bracket to the proper position, the arch may be held in passive adjustment or activated into a power plant. Each banded tooth carries, therefore, a means of applying force to move itself in a mesial or distal direction by a simple adjustment of the bracket, and, at the will of the operator, teeth can be activated to move en masse in a mesial or distal direction by correlating the action of the adjustable locking brackets.

The arch wire is flexed by the adjusted bracket to provide the desired power for tooth movements. This is contrary to the present technique wherein force is applied by making a bend or a series of bends, known as second order bends, in the arch wire which is inserted into fixed bracket channels and tied with ligatures to move teeth.

Now, instead of bending the arch wire, the bracket may be adjusted to change the angulation of one bracket channel, or the channels of a series of brackets, so that they are parallel to each other, thus making it possible to

place the arch wire, free from bends in the gingivo-occlusal direction, into the bracket channels.

The arch wire is locked without the use of ligatures, pins, etc., and is in engagement with the bracket channels automatically when the bracket is turned at a slight angle to the long axis of the tooth, and the arch will not disengage itself during treatment (Fig. 2).

It can be readily seen that second order bends, the complexities involved in placing exact angulation in the bends, and the tedious time-consuming task of reproducing exactly the bends of one buccal segment in the buccal segment on the opposite side of the arch wire, are eliminated.

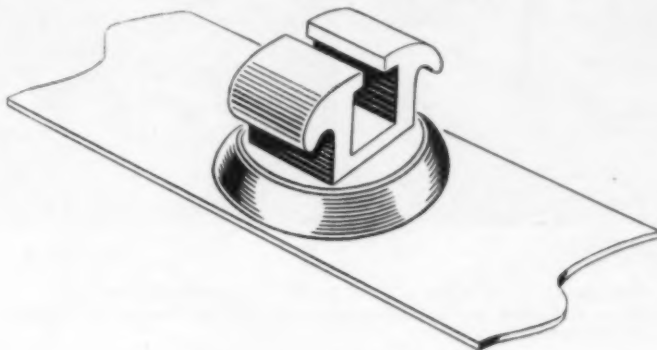


Fig. 1.—Adjustable bracket.

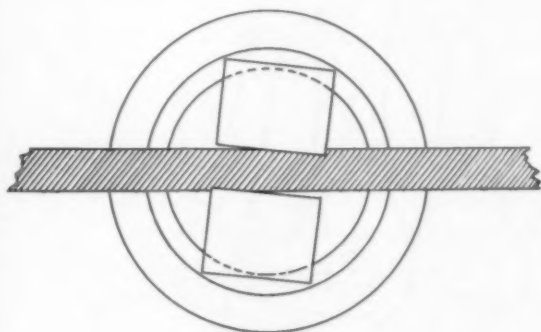


Fig. 2.

Fig. 2.—Adjustable bracket locking arch wire.

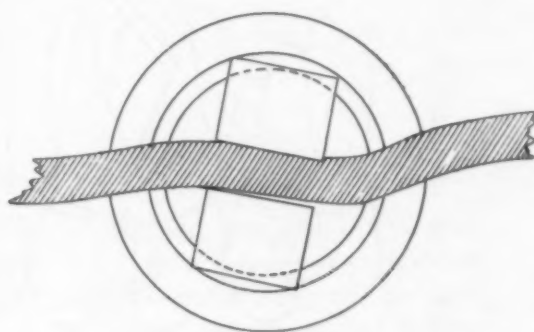


Fig. 3.

Fig. 3.—Adjustable bracket rotated for tipping action.

The changing of arches from the light resilient working arch to the heavier, stabilizing arch is greatly simplified. Duplicating arches in treatment is made easier, and the complexities of forming, inserting, and controlling an arch wire embodying both second and third order bends are eliminated because only the third order bends, or torque force, is now incorporated in the arch wire.

To show in detail the advantages and properties of this new device, the present technique in edgewise arch manipulations will be reviewed. This paper will not attempt, in its limits, to cover in detail the multiple points and precise technical procedures described in textbooks on the treatment of malocclusion by the edgewise technique. Its purpose will be to review and outline the edgewise arch technique and to indicate the changes in the technique introduced by the Adjustable Locking Bracket.

BANDING THE TEETH

The present technique requires that bands be placed on the teeth with the channel of the bracket at right angles to the long axis of the teeth in malposed positions.

The Adjustable Locking Bracket permits the correction of any faults in the angulation of the bracket channels so that, at the inception of treatment, each slot will be at right angles to the long axis of the tooth upon which it is placed regardless of its degree of malposition, and be in the correct position to begin the "leveling off" process (Figs. 4 and 5).



Fig. 4.



Fig. 5.

Fig. 4.—Bracket channels at right angles to the long axis of the teeth.

Fig. 5.—Brackets adjusted for distal movement of the buccal segments. Wings of the brackets are parallel to each other.

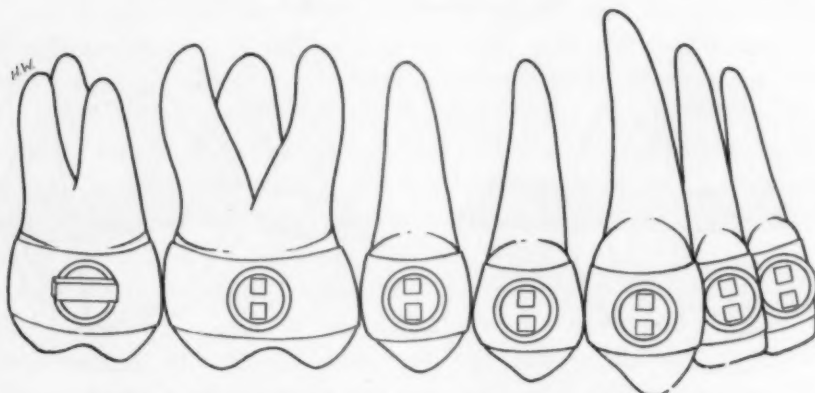


Fig. 6.—Proper band and bracket location on upper teeth.

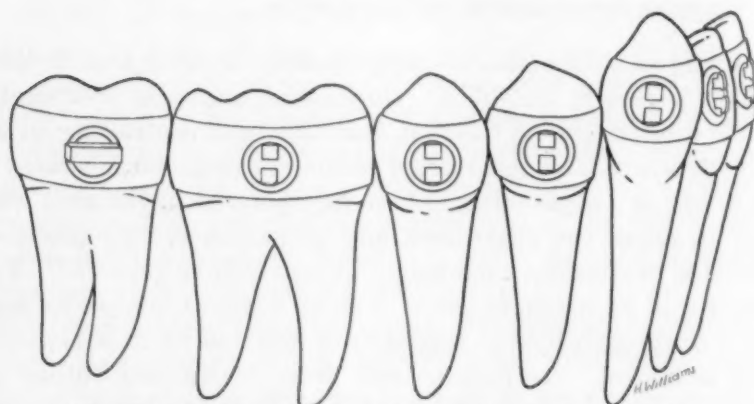


Fig. 7.—Proper band and bracket location on lower teeth.

The brackets are centered mesiodistally, if possible, on the labial and buccal surfaces of the teeth, whether they require rotation or not (Figs. 6 and 7). In the case where proper location of the bracket is impossible because of rotational malocclusion, cusp interference, or incisors blocked out of alignment, etc.,

the brackets are placed as close to the proper position as possible, and, later, after the interference is eliminated, the band is removed and reshaped to conform to the ideal bracket location and then recemented.

On upper anchor molars, the gingivo-occlusal position of the adjustable tube should be in the center of the tooth, and on lower molars slightly gingival to the center, if conditions will permit.

On anchor molars, the tube can be adjusted so that the channel lies parallel to the occlusal plane and at right angles to the long axis of the tooth. On badly tipped molars, the tube is adjusted to accept the arch wire more easily than it is received in the present technique, and the tube channel is adjusted progressively as judgment dictates until the molar is upright and in normal position (Fig. 8).

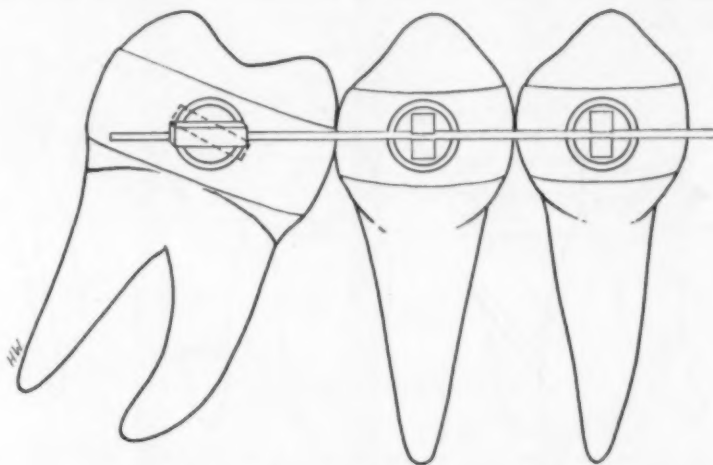


Fig. 8.—The uprighting of the molar.

Anchor molars in excessive rotational malocclusion may require a compensatory buccal or lingual bend in the arch wire, before bracket engagement is possible, but these will never require a gingivo-occlusal bend in the arch wire, no matter how severe the tipping of the molar, because the adjustable tube, as already mentioned, eliminates the necessity for these bends.

PRELIMINARY STAGES OF TREATMENT, OR THE "LEVELING OFF" PROCESS

The arch wires are formed as in the present technique. A round wire, 0.016 or 0.018 inch, is used, depending upon the degree of divergence of the malposed teeth from the horizontal plane, and upon the spaces in the denture caused by unerupted teeth.

The wire is given very little arch form. Molar stops are fashioned with Nance pliers or No. 139 pliers. The arch is tested by placing it on a glass slab where it should lie flat. The adjustable tube on the anchor molar is turned gingivally to a slight degree so that when the arch is inserted the incisor segment of the arch wire will lie gingivally to the incisor brackets. The arch wire is first placed into the incisor bracket channels with finger pressure and then placed into the channels of the brackets of the buccal segments (Fig. 9). Where it is impossible to obtain bracket engagement because of extreme vertical divergence of malposed teeth, ligatures are used until bracket engagement is found possible at subsequent appointments.

The tooth movements produced are limited to changes in vertical positions, because the object is to bring the bracket channels into the same horizontal plane. There is little or no lateral expansion in the molar areas, and only slight lateral expansion in the premolar and cuspid areas.

The object of this preliminary stage of treatment is to bring each tooth into a harmonious vertical position, with the mesiodistal axial inclination of the teeth parallel with each other or as nearly parallel as possible, thus bringing the bracket location on each tooth

to the same horizontal plane. After complete bracket engagement is obtained, with the 0.016 or 0.018 inch round wire, new arches are formed using 0.020 inch round wire.

These new arches are shaped to the working arch form, using the patient's predetermined arch form as a guide. Arch charts of the upper and lower denture are used constantly in the forming of arches (Fig. 10.)

In shaping the 0.020 inch round wire, it is desirable to incorporate greater flattening of the incisor area in both the maxillary and mandibular arches, and positive lateral expansion in the buccal segments.

Loops are incorporated in the arch wire for tying to the anchor molar. The arch wire is tested for symmetry on the patient's chart, and for horizontal plane levelness on the glass slab.

At this stage of the preliminary treatment, before placing the arch wire into the bracket channels, adjustments are made so that any tooth or teeth in greater axial malposition than the other teeth in contact with the arch may be uprighted to harmonious axial inclination (Fig. 5).

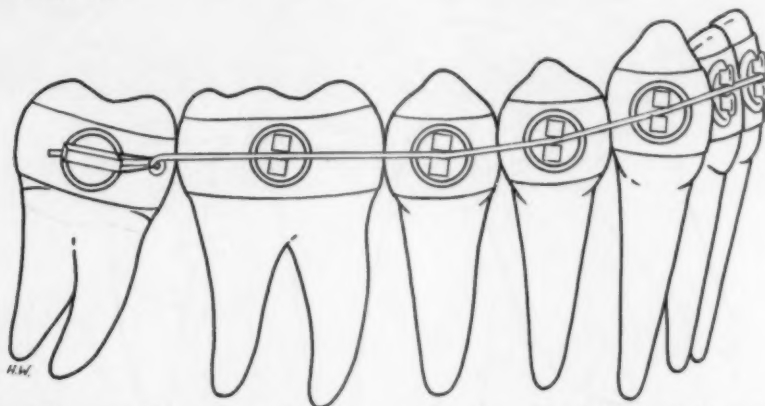


Fig. 9.—The assembly with small gauge round wires. Brackets in locking position.

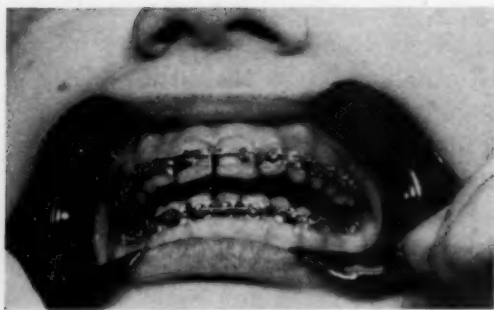


Fig. 10.

Fig. 10.—Band assembly in the mouth.



Fig. 11.

Fig. 11.—Brackets adjusted for locking of the arch wire—0.020 inch round wire in assembly.

The anchor molar tubes are adjusted to increase the distal tipping of the anchor teeth slightly. The bracket channels of the premolar and cuspid teeth are adjusted to lock the arch wire sufficiently to prevent displacement. *No adjustment of brackets for tooth movement should be attempted after insertion of the arch wire into the bracket channels.*

When placing 0.020 inch round wires into the mouth, the arch wire is inserted into the molar tubes and then with finger pressure into the brackets of the incisors. The premolar and cuspid brackets are the last engaged. The arch wire is ligated to the anchor molars. (Figs. 10 and 11.)

The adjustments of the brackets mentioned are made by using a close-fitting wrench or key (Fig. 12). When *distal* movement of a crown is desired, the *occlusal wing* of the

bracket is turned *mesially*. When *mesial* movement of a crown is desired, the *occlusal wing* of the bracket is turned *distally*.

Slight rotation of the bracket channel from positions parallel to the arch wire locks it in position without exerting force on the tooth (See Fig. 2). Further movements produce frictional pressure on the arch wire which may be increased until definite and positive force is in evidence (See Fig. 3). In the preliminary stages of treatment, care must be taken in making adjustments so that the pressures applied on the arch wire will upright the teeth into a harmonious axial relationship without bringing about severe tipping and excessive movement of the roots.

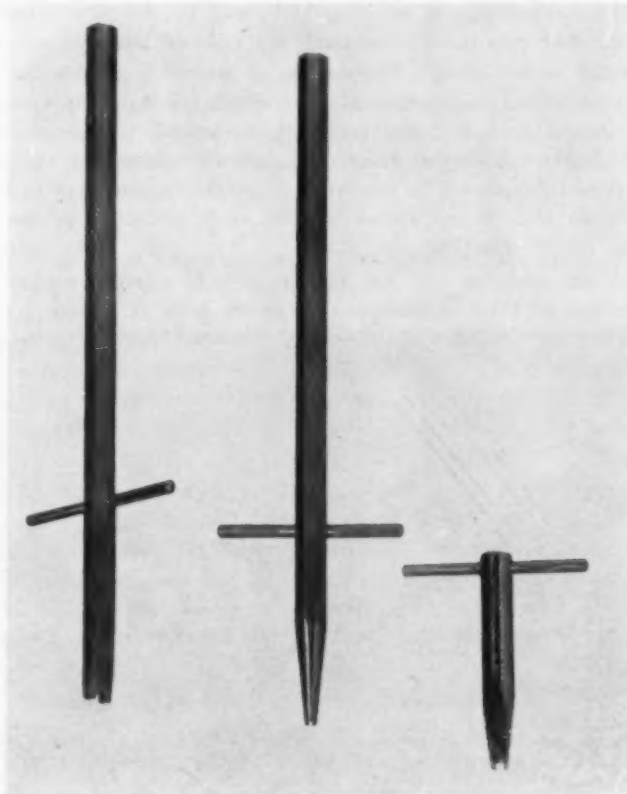


Fig. 13.

Fig. 12.

Fig. 12.—Keys for adjusting of brackets.

Fig. 13.—Torquing iron for insertion of arch wire into bracket channels.

The preliminary stages of treatment are confined to reshaping the denture and bringing the arches to that form wherein they will be most receptive to the placement of rectangular arch wires. The 0.016 and 0.018 inch round wires are used only for "leveling off" of the dentures, and very little expansion is obtained with them. The 0.020 inch round wire which follows them is used: first, to obtain lateral expansion within sound physiologic concepts; second, to reshape the contour of the incisor segments into a flat or less convex outline; third, to upright the badly malpositioned teeth into a harmonious relationship with adjacent teeth; fourth, to bring all bracket channels into the same horizontal plane.

The distal and mesial tipping of teeth and the expansion required for normal arch form and size as based on patients' charts are obtained with rectangular arches.

Intermaxillary elastics alone or in combination with occipital anchorage are normally used only with the rectangular arches. The use of intermaxillary elastics with 0.020 inch round wires is not advocated, but, in the few cases

when it is desirable to make use of them, the mesial or distal pressures on the arch wire may be intensified to assist in producing movement of the segments in the desired direction.

RECTANGULAR ARCH WIRE THERAPY

In continuing the treatment with rectangular arches, a resilient or working arch wire, 0.021 by 0.025 inch, is shaped and formed for the lower arch, using the patient's predetermined arch chart. The technique followed is the same as in the present edgewise arch technique with few exceptions. The arch wire is inserted into the mouth, marked for length, and the bracket markings of all the teeth, and of the mesial ends of the anchor molar tubes, are scratched into it. If practical, the ends of the arch are extended $\frac{1}{8}$ inch beyond the end of the molar tubes. The degree of lateral expansion in the arch wire is noted and adjusted to obtain an expansion force within biologic concepts. Flatness is incorporated in the incisor area and the necessary horizontal torque placed in the buccal segments. A mild degree of lingual torque is normally placed in the incisors area, although no torque should be placed in this area when the incisors are in marked labial inclination. It is desired that torque forces be held to a minimum in the rectangular arch wire therapy at the initial insertion.

Lingual bends are made at the end of the arch to prevent anchor molar rotations. A slight lingual horizontal bend is incorporated in the arch at a point corresponding with the mesial surface of the first molar. The arch is tested for symmetry on the patient's chart and for horizontal plane levelness. The arch wire is first inserted into the anchor tubes and then placed into the incisor brackets with finger pressure and in the buccal segments with the use of the torquing iron (Fig. 13). The arch wire is ligated at the anchor molar tubes.

At the next appointment, the working arch is adjusted to provide the needed progressive torquing force in the buccal segments, and lingual incisal torque if the teeth are not in too great labial protrusion. The incorporation of distal mass movements of the buccal segments is made by the precise adjustment of the brackets, the wings of all brackets being kept practically parallel to each other. The molar and anchor molar brackets are adjusted to a slightly greater angle to the long axis of these teeth, and these are parallel to each other (Fig. 14).

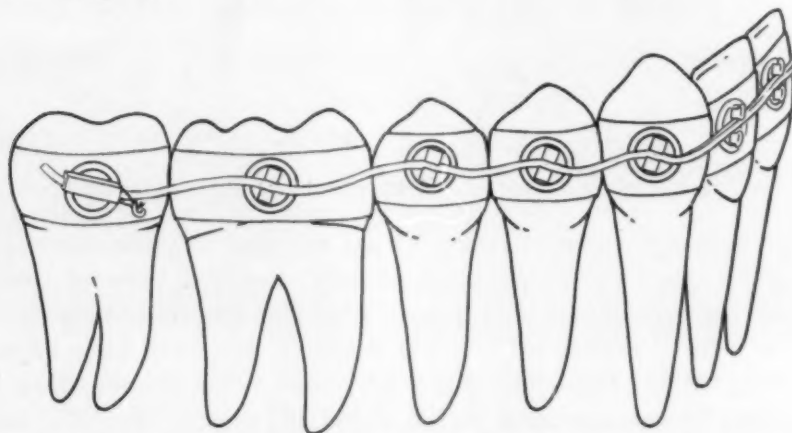


Fig. 14.—Bracket position for distal movement.

In returning to its original state or straight line form, the arch wire will move the teeth and produce our objectives, namely, the uprighting and distal movement of the buccal segments, with a lingual inclination of the buccal segments.

The incisors will have lingual pressure exerted and will be inclined to lingual tipping (Fig. 15).

The above therapy is applied in conjunction with intermaxillary elastics alone or in combination with occipital anchorage.

On the upper arch, a heavy stabilizing wire, 0.022 by 0.028 inch, is formed and placed while dynamic stationary anchorage is being obtained in the lower arch, as in the present edgewise arch technique (Fig. 16). Slight distal tip-back movement is obtained by the adjusting of the brackets, and the arch wire is inserted as previously described in the application of the lower arch (Fig. 17).

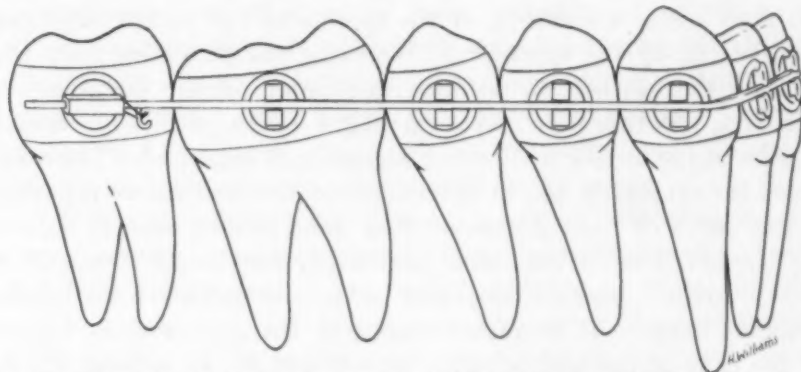


Fig. 15.—Dynamic stationary anchorage established in the mandibular denture.

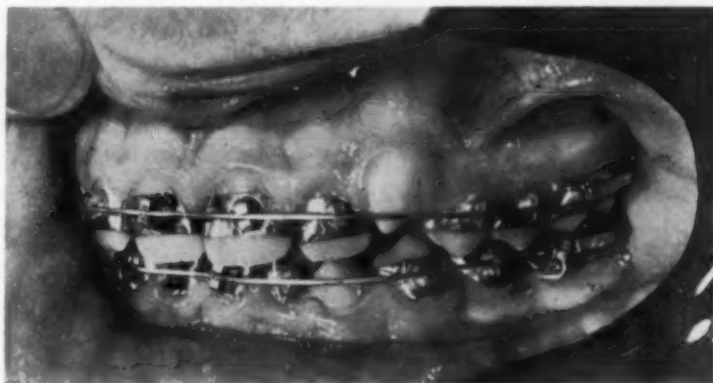


Fig. 16.—Brackets adjusted for tip-back movements of buccal segments and locking of the arch wire in incisor segments. Ligature ties at anchor molar.

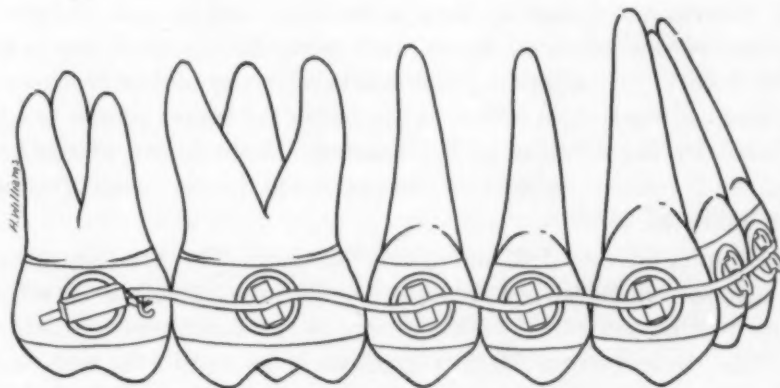


Fig. 17.—Arch wire activated by rotation of brackets.

To review briefly: In preparing the mouth for the insertion of the arch, the brackets on the molars, premolars and cuspids are adjusted to incorporate mild distal mass movements of the buccal segments, and their channels are practically parallel to each other. The molar brackets are adjusted to a slightly greater degree of angulation than the premolar and cuspid brackets. The arch wire is placed into the incisor brackets with finger pressure and into buccal

segment brackets with the torquing iron. No adjustments of the incisor brackets are made at this time unless root and crown movements are desired. If crown and root movements are required, the brackets on those teeth requiring movement are adjusted as indicated in a mesial or distal direction. During the treatment periods while employing elastic ligatures for intermaxillary force, the mass distal movements of the crowns of the buccal teeth can be increased as desired by the operator by removing the arch wire from the mouth and adjusting the brackets to produce an increase in the distal movement of these segments. At this time increased torque action can be incorporated into the arch wire in the incisor and buccal segments, if indicated. The arch wire is again tested for symmetry and horizontal plane levelness before replacing in the mouth. The curve of Spee is incorporated into the arch wire if indicated. In adjusting the brackets for increased mass distal movements it is well to follow a cautious procedure, and the increases in the angulation of the bracket channels should be slight. If increased length of the arch wire is indicated, the spurs at the ends of the arch wire are moved distally to increase the length of the arch from anchor molar tube to anchor molar tube. The lingual torque force on the incisors should always be of a less degree than the buccal segment tipping force, otherwise the incisor segment will act as a brake and retard the distal movement of the buccal segments. The desired objective is the movement of the dentures as a unit, the combined forces embodied within the arch wire supplemented by the elastic forces producing the desired objective.

It was previously stated that no torque force was placed in the incisor segments when the incisor segments are in labial protrusion, the reason being that torque action is already in force when the arch wire is inserted into the bracket channels of the incisors due to the divergence from the horizontal planes of the bracket channels on labially protruded teeth. After the labial protrusion is reduced, the torque action is increased by placing lingual torque in the arch wire. (See Fig. 15.) It was also stated previously that on labially protruded incisors the brackets are not adjusted to exert force on the arch, the reason being that a loose engagement is desired, otherwise a force is set up which acts as a brake and retards the lingual movement of these teeth. When the labial protrusion of the incisors is reduced and lingual torque force is placed in the incisor segment of the arch wire, the brackets are adjusted to lock the arch wire. In adjusting the brackets of the incisor teeth to lock the arch wire and convey torque action to the teeth, the lumen of the bracket channel is reduced by the rotation of the bracket. Adjustment of the bracket on the lateral and central incisors is always made in the desired direction of movement indicated.

For example, in crowded and rotated incisors, the brackets are adjusted with the occlusal wings turned mesially so that the direction of movement of the incisors will be controlled and conform to the forces embodied in the arch wire. In other words, when lateral expansion is desired in the incisor area, and the arch wire embodies that expansion, the brackets are adjusted to obtain distal movement of the incisors and the forces are coordinated. It has been proved by clinical experience that when the arch wire embodies lateral expansion in the incisor area, but the brackets were adjusted on the incisors to obtain mesial movement of the incisors, the force inserted in the arch wire by the bracket adjustment is greater than the force inherent in the arch wire for lateral expansion and the two forces are in conflict, one acting as a brake against the other. It is most important that the two sources of power be in

harmony and work in coordination, namely, the force inherent in the arch wire, and, second, the force inserted in the arch wire by the adjusted bracket. Of the two, the latter is the greater and stronger force and will predominate when not in harmony.

During this period of treatment wherein the tip-back action and progressive torque force are in action on the buccal segments, it is well to remember that both adjustments must be simultaneously incorporated and coordinated on these teeth. In other words, progressive torque force should not be incorporated in an arch wire unless the angulation of the brackets is adjusted for distal movement of the tooth in combination with the elastic forces. In the present technique, some changes are required in the length of the interbracket segments. As the buccal teeth tip distally, the short interbracket segments of the arch wire, between the first and second molars and between the second premolars and first molars, must be increased in length or distal tipping of the molars will be retarded. This is necessary because of the greater mesiodistal length of the molars, which causes their mesial portion to elevate to a greater degree and their distal portion to be depressed more extensively than the corresponding tooth areas in the premolars. In the technique now described, the molar brackets are adjusted so that the angulation of the bracket channels are of a slightly greater degree than that in the premolar area, and the distal tipping actions are thus coordinated.

There are many points of interest in the application of the Adjustable Locking Bracket that have not been mentioned. These are of such a nature that in working with the appliances they will be promptly recognized. A few of these points are:

1. The use of the Adjustable Locking Bracket will give the better balance between functional and mechanical stresses.

2. The bracket engagement can be rigid or nonrigid. Bracket engagement that is positive in its control, yet not so binding that it interferes with functional stresses, is ideal; for example: when the bracket is adjusted to tip the crown of a tooth distally, the apparent opening of the bracket channel is reduced and the working points of the bracket channel in contact with the arch wire can be set very rigidly, and as the tooth is tipped into its new position by the mechanical force and the vertical axis of the tooth is changed, the tooth automatically changes its relationship with the arch wire and the bracket engagement changes into a nonrigid type. The tooth is held in its new position, yet the mechanical force incorporated by the bracket adjustment is greatly reduced and the functional forces are in command.

3. Treatment with this type of adjustable locking bracket will require fewer manipulations and fewer appointment periods because the arch wires and brackets will be left without interference until the forces applied by adjustment of the brackets have had their maximum effect reduced.

Dr. Strang¹ has written "that the prime motive that stimulated Dr. Angle to continue his efforts to produce a more efficient mechanism, subsequent to the introduction of the Ribbon Arch Appliance, was the realization that teeth must be moved distally in the majority of cases of malocclusion, if the normal was to be obtained in treatment. The second objective was to furnish an appliance by means of which greater stability of anchorage units could be evolved. His answer to these two problems was the Edgewise Arch Mechanism." These objectives may be reached more efficiently through the use of the Adjustable Locking Bracket.

There is no appliance in the orthodontist's armamentarium from which he can obtain so great an amount of anchorage with the use of a *single arch wire* as he can now obtain by means of this technique. Anchorage is the pillar upon which the success of orthodontics is based. Without adequate anchorage, the successful completion of an orthodontic case is impossible.

The use of the Adjustable Locking Bracket in combination with the edge-wise arch greatly enhances the normal anchorage, and many treatment complexities are eliminated. Anchorage with this new device may be obtained in as many single or multiple units as is desired. The dynamics and physiology in tooth movements will be in better balance. The forces employed are positive and directionally controlled with the expenditure of a minimum of chair time. Fewer appointments are necessary and inconvenience to the operator and to patients is minimized.

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Department of Orthodontic Abstracts and Reviews

Edited by

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The Fels Composite Sheet. I: A Practical Method for Analyzing Growth Progress: By Lester W. Sontag, M.D., and Earle L. Reynolds, Ph.D., Yellow Springs, Ohio, *J. Pediat.* 26: 327-335, April, 1945.

Weight is probably the most common figure available. Height is easily obtained. Occasionally an x-ray may be taken to evaluate the state of skeletal growth and maturation. Time of eruption of teeth is a maturational factor not usually considered of great importance, but at times it is of significance, and it is often of concern to the mother. This paper presents, in the form of a composite sheet, a methodology for recording and interpreting four different items of growth data, together with the norms for their use.

TABLE I. HEIGHT (CM.)*

| AGE | BOYS | | GIRLS | |
|--------|-------|--------------------|-------|--------------------|
| | MEAN | STANDARD DEVIATION | MEAN | STANDARD DEVIATION |
| Mo. B | 50.1 | 2.5 | 49.6 | 2.0 |
| 1 | 54.0 | 1.8 | 54.0 | 1.8 |
| 3 | 60.7 | 2.0 | 59.2 | 2.0 |
| 6 | 67.5 | 2.4 | 66.0 | 2.0 |
| 9 | 72.0 | 2.2 | 70.5 | 2.0 |
| 12 | 75.5 | 2.3 | 74.2 | 2.3 |
| Yr. 1½ | 81.7 | 2.5 | 80.8 | 2.5 |
| 2 | 87.2 | 2.9 | 86.2 | 2.7 |
| 2½ | 91.8 | 3.0 | 90.6 | 3.2 |
| 3 | 95.7 | 3.4 | 94.8 | 3.4 |
| 3½ | 99.7 | 3.7 | 98.5 | 3.5 |
| 4 | 103.2 | 4.1 | 102.0 | 3.8 |
| 4½ | 106.7 | 4.5 | 105.2 | 4.1 |
| 5 | 110.2 | 4.6 | 109.0 | 4.1 |
| 5½ | 113.7 | 4.8 | 112.4 | 4.4 |
| 6 | 117.2 | 4.9 | 115.7 | 4.5 |
| 6½ | 119.4 | 4.5 | 118.2 | 4.7 |
| 7 | 122.0 | 4.8 | 120.9 | 4.9 |
| 7½ | 125.1 | 5.0 | 124.4 | 5.1 |
| 8 | 127.7 | 5.1 | 126.9 | 5.4 |
| 8½ | 130.9 | 5.1 | 129.4 | 5.0 |
| 9 | 133.7 | 5.4 | 133.1 | 6.1 |
| 9½ | 136.7 | 5.6 | 136.0 | 6.0 |
| 10 | 140.0 | 5.8 | 139.4 | 6.6 |
| 10½ | 142.2 | 6.0 | 142.0 | 6.8 |
| 11 | 145.0 | 6.1 | 145.1 | 7.1 |
| 11½ | 147.2 | 6.5 | 148.9 | 7.3 |
| 12 | 149.9 | 6.9 | 151.9 | 7.6 |
| 12½ | 152.8 | 7.4 | 154.8 | 7.1 |
| 13 | 155.8 | 7.9 | 157.0 | 6.6 |
| 13½ | 158.1 | 8.0 | 159.0 | 6.2 |
| 14 | 161.7 | 8.1 | 160.4 | 5.8 |
| 14½ | 164.4 | 8.2 | 161.5 | 5.5 |
| 15 | 167.5 | 8.4 | 162.4 | 5.3 |
| 15½ | 170.2 | 8.0 | 163.0 | 5.2 |
| 16 | 172.2 | 7.6 | 163.4 | 5.1 |
| 16½ | 173.9 | 7.1 | 163.4 | 5.0 |
| 17 | 174.7 | 6.6 | 163.1 | 4.8 |

*Values through 9½ years are Fels norms. Older age levels adapted from Richey, by permission.

The group of children were under observation at the Fels Research Institute. Serial measurements of height, weight, appearance of teeth, and ossification centers in approximately two hundred and fifty children have been the basis for the norms used in the Fels growth composite sheet. The subjects are normal white children, they come from families of average or better than average economic status, and have been enrolled in the study since birth.

The composite sheet is designed to permit the recording of any growth item such as height, weight, ossification, or dentition at any age at which a

TABLE II. WEIGHT (Kg.)

| AGE | BOYS | | GIRLS | |
|--------|------|--------------------|-------|--------------------|
| | MEAN | STANDARD DEVIATION | MEAN | STANDARD DEVIATION |
| Mo. B | 3.5 | .5 | 3.4 | .5 |
| 1 | 4.2 | .6 | 4.0 | .5 |
| 3 | 5.9 | .7 | 5.4 | .6 |
| 6 | 7.8 | .9 | 7.1 | .7 |
| 9 | 9.1 | .9 | 8.5 | .8 |
| 12 | 10.0 | .9 | 9.5 | .8 |
| Yr. 1½ | 11.4 | 1.0 | 10.8 | 1.0 |
| 2 | 12.6 | 1.1 | 11.9 | 1.2 |
| 2½ | 13.6 | 1.2 | 12.9 | 1.4 |
| 3 | 14.6 | 1.3 | 13.9 | 1.6 |
| 3½ | 15.6 | 1.4 | 14.9 | 1.7 |
| 4 | 16.6 | 1.6 | 15.9 | 1.8 |
| 4½ | 17.7 | 1.7 | 16.9 | 2.0 |
| 5 | 18.8 | 2.0 | 18.0 | 2.1 |
| 5½ | 20.1 | 2.1 | 19.1 | 2.3 |
| 6 | 21.4 | 2.2 | 20.4 | 2.5 |
| 6½ | 22.5 | 2.5 | 21.1 | 2.6 |
| 7 | 23.7 | 2.9 | 22.4 | 3.1 |
| 7½ | 25.2 | 3.3 | 24.0 | 3.5 |
| 8 | 26.4 | 3.4 | 25.2 | 3.8 |
| 8½ | 28.4 | 4.0 | 26.8 | 4.2 |
| 9 | 30.3 | 4.4 | 28.6 | 4.7 |
| 9½ | 31.8 | 4.9 | 30.7 | 5.7 |
| 10 | 33.0 | 4.5 | 33.0 | 5.9 |
| 10½ | 34.0 | 4.8 | 35.6 | 6.2 |
| 11 | 35.7 | 5.2 | 38.0 | 6.6 |
| 11½ | 37.5 | 5.8 | 40.2 | 7.0 |
| 12 | 39.3 | 6.4 | 42.6 | 7.3 |
| 12½ | 41.5 | 6.8 | 44.9 | 7.6 |
| 13 | 43.8 | 7.3 | 47.4 | 7.9 |
| 13½ | 46.1 | 7.5 | 49.3 | 7.8 |
| 14 | 48.5 | 7.7 | 50.9 | 7.7 |
| 14½ | 51.0 | 7.8 | 52.0 | 7.3 |
| 15 | 53.4 | 7.9 | 53.4 | 7.0 |
| 15½ | 56.3 | 8.1 | 54.3 | 7.0 |
| 16 | 57.9 | 8.2 | 54.9 | 6.9 |
| 16½ | 59.3 | 7.7 | 55.0 | 6.8 |
| 17 | 61.3 | 7.3 | 55.0 | 6.8 |

TABLE III. NUMBER OF OSSIFICATION CENTERS PRESENT

| AGE IN MO. | BOYS | | GIRLS | |
|------------|------|------|-------|------|
| | M. | S.D. | M. | S.D. |
| 1 | 4.2 | 1.2 | 4.7 | 1.8 |
| 3 | 6.4 | 1.6 | 7.7 | 2.0 |
| 6 | 9.7 | 2.0 | 10.9 | 2.9 |
| 9 | 11.4 | 2.6 | 15.0 | 4.7 |
| 12 | 13.7 | 4.2 | 21.0 | 6.0 |
| 18 | 20.8 | 6.7 | 32.7 | 6.8 |
| 24 | 28.2 | 7.1 | 40.8 | 6.3 |
| 30 | 35.4 | 6.6 | 46.0 | 5.5 |
| 36 | 41.3 | 5.7 | 50.5 | 4.7 |
| 42 | 44.4 | 5.4 | 53.9 | 3.5 |
| 48 | 48.2 | 5.1 | 55.7 | 2.9 |
| 54 | 50.9 | 4.5 | 56.6 | 2.6 |
| 60 | 53.2 | 4.0 | 58.6 | 2.1 |
| 66 | 54.7 | 3.5 | 59.3 | 1.9 |
| 72 | 56.4 | 3.1 | 60.0 | 1.8 |
| 78 | 57.3 | 2.9 | 60.4 | 1.5 |

child is measured, and in terms of its position in relation to the Fels group of normal children.

Every measurement, whether it be height, weight, or other growth phenomenon, can, from the proper table of norms, be converted into an expression of the degree to which it deviates from the mean. This value can be

TABLE IV. TIME OF APPEARANCE OF INDIVIDUAL OSSIFICATION CENTERS

| EPIPHYSES AND CENTERS OF OSSIFICATION | AGE WHEN OSSIFICATION BEGINS | | | |
|--|------------------------------|------|--------------|------|
| | BOYS | | GIRLS | |
| | M. MONTHS | S.D. | M. MONTHS | S.D. |
| 1 Distal femur | 0 | | 0 | |
| 2 Proximal tibia | 0.1 | | 0.1 | |
| 3 Cuboid | 0.5 | | 0.4 | |
| 4 Head of humerus | 0.7 | | 0.9 | |
| 5 Capitate | 2.4 | 1.8 | 2.3 | 2.1 |
| 6 Hamate | 3.4 | 2.2 | 2.5 | 2.3 |
| 7 Distal tibia | 3.9 | 1.5 | 3.4 | 1.4 |
| 8 Head of femur | 4.4 | 2.0 | 3.7 | 1.6 |
| 9 Lateral cuneiform | 4.4 | 4.3 | 3.8 | 4.4 |
| 10 Capitulum | 6.3 | 4.3 | 4.1 | 3.6 |
| 11 Gt. tuber. humerus | 11.4 | 7.2 | 6.6 | 3.3 |
| 12 Distal fibula | 12.5 | 4.1 | 9.3 | 2.6 |
| 13 Distal radius | 13.0 | 4.7 | 10.8 | 4.4 |
| 14 Prox. 3rd finger | 16.2 | 5.3 | 10.4 | 3.1 |
| 15 Distal 1st toe | 16.8 | 5.6 | 10.6 | 2.8 |
| 16 Prox. 2nd finger | 17.3 | 5.0 | 11.0 | 3.0 |
| 17 Prox. 4th finger | 17.7 | 5.4 | 11.1 | 3.2 |
| 18 Metacarpal II | 17.9 | 5.1 | 12.8 | 3.7 |
| 19 Distal 1st finger | 18.4 | 6.2 | 12.8 | 5.0 |
| 20 Prox. 3rd toe | 19.5 | 5.2 | 12.2 | 3.8 |
| 21 Prox. 4th toe | 21.0 | 5.1 | 13.6 | 3.8 |
| 22 Metacarpal III | 21.1 | 6.4 | 14.2 | 4.0 |
| 23 Medial cuneiform | 21.9 | 9.9 | 16.7 | 8.5 |
| 24 Prox. 5th finger | 22.2 | 5.6 | 15.2 | 4.2 |
| 25 Prox. 2nd toe | 22.2 | 5.8 | 14.1 | 3.8 |
| 26 Metacarpal IV | 23.6 | 7.1 | 16.0 | 4.1 |
| 27 Middle 3rd finger | 24.9 | 7.6 | 15.9 | 4.9 |
| 28 Middle 4th finger | 24.9 | 7.8 | 15.8 | 4.8 |
| 29 Metacarpal V | 26.0 | 8.0 | 17.2 | 4.7 |
| 30 Middle 2nd finger | 26.9 | 7.5 | 17.3 | 5.2 |
| 31 Triquetral | 27.3 | 15.9 | 23.6 | 13.7 |
| 32 Metatarsal I | 27.7 | 4.7 | 20.1 | 3.3 |
| 33 Distal 3rd finger | 27.8 | 6.4 | 20.2 | 3.9 |
| 34 Distal 4th finger | 28.3 | 7.0 | 19.9 | 5.9 |
| 35 Middle cuneiform | 28.4 | 11.2 | 21.3 | 7.6 |
| 36 Metacarpal I | 29.8 | 7.3 | 20.3 | 5.3 |
| 37 Prox. 1st toe | 29.9 | 5.8 | 20.3 | 5.5 |
| 38 Prox. 5th toe | 32.0 | 5.9 | 21.3 | 4.8 |
| 39 Navicular foot | 33.4 | 13.5 | 25.8 | 11.1 |
| 40 Metatarsal II | 33.4 | 6.8 | 25.8 | 6.1 |
| 41 Prox. 1st finger | 34.8 | 7.9 | 21.6 | 5.1 |
| 42 Distal 2nd finger | 37.0 | 7.9 | 25.8 | 6.9 |
| 43 Distal 5th finger | 37.4 | 7.4 | 25.5 | 7.0 |
| 44 Middle 5th finger | 40.3 | 11.7 | 24.9 | 7.9 |
| 45 Metatarsal III | 41.5 | 7.9 | 29.1 | 6.4 |
| 46 Gt. troch. femur | 42.6 | 7.6 | 29.8 | 6.4 |
| 47 Lunate | 46.0 | 19.3 | 34.6 | 14.2 |
| 48 Proximal fibula | 47.0 | 11.8 | 32.6 | 9.3 |
| 49 Metatarsal IV | 48.7 | 9.0 | 34.0 | 7.2 |
| 50 Distal 4th toe | 51.2 | 10.1 | 30.7 | 7.9 |
| 51 Patella | 51.9 | 11.6 | 34.8 | 8.5 |
| 52 Distal 3rd toe | 53.5 | 11.2 | 32.8 | 7.7 |
| 53 Metatarsal V | 53.6 | 10.6 | 38.6 | 8.4 |
| 54 Distal 2nd toe | 57.0 | 11.4 | 35.5 | 7.3 |
| 55 Navicular hand | 60.1 | 14.1 | 47.8 | 12.3 |
| 56 Proximal radius | 63.5 | 17.2 | 47.5 | 12.1 |
| 57 Greater multangular | 64.3 | 19.7 | 47.0 | 14.8 |
| 58 Lesser multangular | 64.4 | 15.2 | 48.3 | 14.8 |
| 59 Med. ep. humerus | 73.6 | 17.5 | 41.3 | 9.9 |
| 60 Distal ulna | 82.4 | 10.6 | 63.2 | 15.3 |
| 61 Epiph. calcaneus | 89.6 | 14.0 | 63.7 | 11.8 |
| Time of fusion in first phalanx of index finger | 16.7 | 1.3 | 14.2 | 1.1 |

plotted on the same composite sheet with other types of measures which are also expressed in terms of their distribution in the group.

Any other data for which means and standard deviations are available may be plotted on the composite sheet. Examples are:

1. Dietary intake in various age levels in terms of proteins, calories, thiamine, etc.

2. Hemoglobin and cell counts

3. Basal metabolic rate

4. Age of menarche

5. Intelligence quotient

6. Vital capacity

7. Motor scores, strength tests, measures of athletic performances, etc.

Means and standard deviations for many of these items and for others are available on our Fels group of normal children. Norms of other groups may, of course, be used with equal facility and some of these are reproduced (with permission of their authors) on the backs of Fels Composite Sheets. At the bottom of the composite sheet is a space for indicating illness in such a way that its relation to growth changes may be readily interpreted.

A method is described for plotting children's growth progress in terms of deviation from the group mean. Individual measurements are transcribed by means of a simpler table into standard scores and thus weight, skeletal development, and dentition may be plotted on the same composite sheet. In this way, interrelationships between various measures of growth may be easily seen. We believe the method reveals a great deal of information often over-

TABLE V, A. AGE AT ERUPTION OF INDIVIDUAL DECIDUOUS TEETH (Mo.)

| TEETH | UPPER JAW (CORRESPONDING TEETH) | | | | LOWER JAW (CORRESPONDING TEETH) | | | |
|-----------------|------------------------------------|------|-------|------|------------------------------------|------|-------|------|
| | BOYS | | GIRLS | | BOYS | | GIRLS | |
| | M. | S.D. | M. | S.D. | M. | S.D. | M. | S.D. |
| Central incisor | 9.1 | 1.5 | 9.6 | 2.0 | 7.3 | 1.6 | 7.8 | 2.1 |
| Lateral incisor | 10.4 | 2.4 | 11.9 | 2.7 | 13.0 | 2.8 | 13.8 | 3.6 |
| Canine | 18.9 | 2.7 | 20.1 | 3.2 | 19.3 | 2.9 | 20.2 | 3.4 |
| First premolar | 16.0 | 2.3 | 15.7 | 2.3 | 16.2 | 1.9 | 15.6 | 2.2 |
| Second premolar | 27.6 | 4.4 | 28.4 | 4.3 | 25.9 | 3.8 | 27.1 | 4.2 |

TABLE V, B.

| AGE IN MO. | DECIDUOUS TEETH (NO.) | | | |
|------------|-----------------------|------|-------|------|
| | BOYS | | GIRLS | |
| | M. | S.D. | M. | S.D. |
| 6 | 0.4 | 0.9 | 0.4 | 0.8 |
| 9 | 3.1 | 1.7 | 2.2 | 2.0 |
| 12 | 6.5 | 1.9 | 5.6 | 2.6 |
| 18 | 12.9 | 2.5 | 12.2 | 2.6 |
| 24 | 17.2 | 1.7 | 16.2 | 2.2 |
| 30 | 19.4 | 1.4 | 18.6 | 1.6 |
| 36 | 20.0 | 0.4 | 19.9 | 0.7 |
| 42 | | | 20.0 | 0.0 |

TABLE VI. AGE DISTRIBUTION OF ERUPTION OF CORRESPONDING PERMANENT TEETH (Yr.)*

| TEETH | UPPER JAW (CORRESPONDING TEETH) | | | | LOWER JAW (CORRESPONDING TEETH) | | | |
|-----------------|------------------------------------|------|-------|------|------------------------------------|------|-------|------|
| | BOYS | | GIRLS | | BOYS | | GIRLS | |
| | M. | S.D. | M. | S.D. | M. | S.D. | M. | S.D. |
| Central incisor | 7.49 | 0.75 | 7.20 | 0.75 | 6.50 | 0.74 | 6.19 | 0.70 |
| Lateral incisor | 8.62 | 1.10 | 8.15 | 0.94 | 7.64 | 0.80 | 7.31 | 0.75 |
| Canine | 11.80 | 1.42 | 11.05 | 1.37 | 10.70 | 1.15 | 9.85 | 1.15 |
| First premolar | 10.42 | 1.50 | 10.00 | 1.40 | 10.75 | 1.42 | 10.20 | 1.41 |
| Second premolar | 11.18 | 1.68 | 10.82 | 1.60 | 11.45 | 1.77 | 11.00 | 1.70 |
| First molar | 6.64 | 0.75 | 6.54 | 0.70 | 6.44 | 0.75 | 6.12 | 0.87 |
| Second molar | 12.70 | 1.18 | 12.40 | 1.40 | 12.20 | 1.20 | 11.90 | 1.55 |

*From Klein, Palmer, and Kremer, by permission.

looked when other methods are used. Any growth factor, physiologic characteristic, or measure of performance for which means and standard deviations are available may be so plotted. The composite sheet is, we believe, well adapted to the needs of many pediatricians for maintaining case records on their patients.

The Fels Composite Sheet. II: Variations in Growth Patterns in Health and Disease. By Earle L. Reynolds, Ph.D., and Lester W. Sontag, M.D., Yellow Springs, Ohio, *J. Pediat.* 26: 336-352, April, 1945.

The Fels method involves the use of a printed ruled sheet, on which a given growth factor may be plotted in terms of its deviation from the group mean. It is believed that the use of this method reveals facts about the status and growth progress of a child which might otherwise be missed. It has the interesting advantage of making it possible to plot a number of different types of growth measures on the same chart. In this paper is presented some of the results of a study of the growth of 150 children, research subjects at the Samuel S. Fels Research Institute, using the Fels Composite Sheet described in the previous article.

A child may show considerable variability in growth during the first ten years. He may be tall for his age at one period and short for his age at another. He may be heavy in relation to his height at one age and light in relation to his height at another. The child may lose or gain weight to a considerable degree at any time. We shall, therefore, discuss this matter of degree of consistency of growth pattern and of body type. We have divided the first ten years of life into two periods, the first year and the remaining nine years. While such a division is to a degree arbitrary, there seems to be a good reason for making it. The first year is one of considerable vulnerability for the child; that is, it is more difficult for him to maintain his nutritional state and water balance during a period of illness, and he is more immediately responsive to the quantity and quality of his dietary than at later ages.

For the sake of description and statistical treatment, we have defined as constant a pattern of growth which does not vary in a range greater than one standard deviation during either of the periods which we are considering. If the total range of height during a period, for instance, does not exceed one standard deviation, the child is considered to have a constant pattern of height growth for that period. Body type, to the degree to which it is determined by height-weight relation, has been considered in the same way. We have designated as constant any body type in which the relationship between weight and height does not vary more than one standard deviation during either of the periods under observation.

It is apparent immediately that it is the rule rather than the exception for the growth pattern of children during the first year to be variable. Approximately two-thirds of both boys and girls have variations (a range of greater than one standard deviation) in height, weight, and ossification curves during the first year. It is of interest also to note that the variability of the curves is not much greater during the subsequent nine years than it is during the first year. Girls are slightly more variable in every category except height, both during the first year and during the next nine years. Only in ossification during the first year is the difference large enough to be of possible statistical significance. Variability in body type or height-weight ratio is of approximately the same magnitude as variability in the other categories.

Growth patterns show a great variety of shapes. Some of the more easily described and recognized ones are:

1. Those of children whose height-weight curves are close to each other, and whose body type is consequently "average." Rates of growth may of course vary tremendously so long as the height-weight curves remain in approximately the same relationship. Such children may be large, average, or small in size.
2. Those of children whose height-weight curves do not coincide with each other, but in which the relationship is fairly constant, and who are consequent-

ly either tall for their weight or short for their weight. Such children may also be either large, medium, or small in size.

3. Those of children showing clear-cut and prolonged shifts from one body type to another.

4. Those of children whose body type remains fairly constant, but whose growth rate is either irregularly variable or cyclical.

Growth Patterns and Heredity.—Children who are consistently underweight, or whose growth progress is otherwise aberrant, may be deviant because of illness, malnutrition, or an endocrine dyscrasia. Or the unusual growth pattern may be an inherited one. It is often impossible for the pediatrician to differentiate the underfed child from the one who is physically deviant because of an inherited body type or growth pattern.

R. P. presents the height, weight, and ossification patterns of a child who is consistently described by the examining pediatrician as having "poor turgor," "flabby musculature," and being "poorly developed." "Malnourished," "not robust," and other similar terms are repeatedly used to describe him. The chart shows him to be maintaining a height superiority. His height curve is between 0.5 and 2.5 standard deviations high. His weight curve, on the other hand, is from slightly below average for his age to 2 standard deviations high, but is inadequate for his height. He is, then, a slender "gangly" child, with stringy muscles and thin subcutaneous tissue. Yet he is one who was born a tall, slender infant and who has maintained his body proportions at nearly a constant level throughout his first ten years. This fact suggests that the characteristics of his growth may have been genetically determined. His progress in ossification is excellent.

Examination of his history of illness shows little except that he has only occasional colds, certainly less than is common for a child of his age. From his nutrition record we learn that he was breast fed for ten months. Cereals and vegetables were started at a very early age as a supplement to the breast feeding, and cod-liver oil and tomato juice in ample quantities were started at the end of the first month and continued throughout the first three years. He has had no gastrointestinal disturbance, always retained his food well as an infant, and ate well as a child. This child has never shown any indication of allergy. He has not been subject to chronic colds. His tuberculin tests are negative, and chest plates at yearly intervals have shown a consistently normal heart and lungs.

Here then is a child who refuses to change his body type despite an optimum nutritional regimen and a freedom from illness. As we have indicated, there is not evidence that his apparent lack of robust health and his persistent underweight for height are in any way related to illness or a dietary inadequacy. His rate of growth is, however, rapid. An explanation for this picture is to be found only when we examine his father and mother. While his mother is a tall, well-built woman, his father is a slender, stringy individual with poor muscular development and visceroptosis, who is anything but robust in appearance, yet who has better than average endurance and is rarely ill.

J. E. is a child consistently slightly underweight for height, but who is, in addition, small. Her height and weight curves are characteristic of a badly nourished child who neither grows rapidly in height nor maintains a quite adequate weight for height. Yet this child's illness history is as negative as is R. P.'s. She has hardly lost more than a day or two at school for the past four years. Her history for one of these years states, "not a day of school lost this year." Tuberculin test, basal metabolic rate, and chest films are all normal. J. E.'s younger brother's growth pattern is almost identical to hers. Neither brother nor sister has been a robust eater, but, on the other hand, their diets have been excellently planned and the food available has been of the best. The explanation for J. E.'s growth pattern is evident when one sees her mother, a very tiny woman of extremely slender build. The fact that J. E. maintains an approximately average rate of ossification progress also suggests that an inadequate dietary is not a factor.

These two children have been selected to illustrate the fact that deviations from the usual growth curve do not necessarily mean illness, nor do they mean

malnutrition in the usual sense. Both are probably expressing genetically determined growth urges and body types.

A. F. is one of a pair of twins and consequently is small at birth. Apparently, however, she has the potentialities for rapid growth, and in five and one-half years her curves go from a standard score of -2 and -3 to a standard score of +2. This child has risen from the lowest 1 or 2 per cent in height and weight at birth to a position in the upper 2 or 3 per cent at 5½ years. Her parents are both large, robust people. One sibling's growth curve, that of her twin, is very comparable to A. F.'s.

Reynolds and Sontag, and other investigators, have demonstrated the existence of a seasonal factor in growth, for height, weight, and ossification. According to this work, maximum growth in height was shown in the six months preceding September, while maximum growth in weight occurred in the six months preceding March.

We have presented here the growth curves of height and weight and rate of appearance of ossification centers of a number of children, as plotted in terms of standard scores on the Fels Composite Sheet. We have used these cases to illustrate:

1. The degree to which children's growth patterns tend to be constant in relation to group growth patterns.
2. Examples of the way in which the genetically determined pattern of a child's growth sometimes tends to express itself despite environmental impacts.
3. The effects of acute and chronic illness on growth patterns.
4. The seasonal and cyclic variations in growth patterns which are often seen.
5. The effects in growth pattern of subclinical hypothyroidism.
6. The rapidity of recovery from one type of adverse prenatal environmental toxemia.

It is our belief that plotting growth curves in terms of standard scores has major advantages over other methods. Such a procedure reveals more dramatically the growth progress of a child, his growth response to good or bad environment of nutrition, and his body type and its changes. It also makes possible the plotting of all sorts of developmental or biochemical nutritional data on the same chart. Such developmental charts are readily adaptable to the needs of many pediatricians for plotting the progress of patients.

News of the death of Dr. Albin Oppenheim in Los Angeles, California, on Nov. 20, 1945, has just reached the JOURNAL.

News and Notes

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Southern Society of Orthodontists

The Twenty-Second Annual Meeting of the Southern Society of Orthodontists will be held in Charlotte, North Carolina, on Jan. 27, 28, and 29, 1946, with headquarters at the Hotel Charlotte. LELAND T. DANIEL, Secretary and Treasurer.

Pacific Coast Society of Orthodontists

CENTRAL SECTION

The regular quarterly meeting of the Central Section of The Pacific Coast Society of Orthodontists was held Sept. 11, 1945, at the Alexander Hamilton Hotel, San Francisco, California.

An afternoon, dinner, and evening meeting, with Dr. Hays Nance of Pasadena as guest clinician, was one of the best attended in a long time. Complete with a variety of models, Dr. Nance took the members to "both sides of the fence" in regard to the variations in the mesiodistal diameters of the three teeth distal to the lower lateral incisors in both the deciduous and permanent dentitions.

Members present were: Drs. Thomas Sweet, Ernest Johnson, Earl Lussier, Charles Konigsberg, E. R. Schroeder, Elgin Jackson, Kenneth Stratton, Ray Lussier, Reuben L. Blake, William Smith, Jack Loughridge, Will Sheffer, Seymore Gray, Bernard Matzen, Allen Scott, Kenneth Terwilliger, Clarence Carey, Glendon Terwilliger, W. F. Walsh, Verne V. Smith, Leland Carter, Arthur F. Skaife, Frederick West, Vernon L. Hunt, J. Camp Dean.

Guests present were: Drs. Ray McClinton, Harry S. Thompson, Everett Watkins, Ray Brownell, Fred E. Havrilla, Howard Jan, Arnold H. Wieser, Wendell Wylie, and Mr. H. G. Elsasser.

SOUTHERN SECTION

The quarterly meeting of the Southern Section of The Pacific Coast Society of Orthodontists was held Sept. 28, 1945, at the Ambassador Hotel, Los Angeles, California. The program was as follows:

2:00 P.M. "The Influence of the Muscles of Mastication and Deglutition on the Development (Expansion) of the Oral and Nasal Cavities." Dr. J. Walter Reeves.

3:00 P.M. "The Practical Application of Biologic Forces in Orthodontic Treatment." Dr. Albin Oppenheim. Dr. Oppenheim showed that with heavy pressure bone cells are killed and treatment is retarded.

4:30 P.M. Round table discussions of interesting and difficult cases.
Table Chairmen:

| | |
|---------------------|-------------------|
| Dr. Robert Gawley | Dr. George Graser |
| Dr. Herbert Shannon | Dr. John Wilson |

The business meeting followed, with Chairman John Abel presiding. Dr. James McCoy gave a discussion of the policies of the AMERICAN JOURNAL OF ORTHODONTICS.

There followed an active Round Table Discussion of the subject, "At What Age Shall I Treat Orthodontic Anomalies?" under the leadership of Dr. McCauley. Questions pertinent to the subject were discussed by Drs. Fahrney, Fluhrer, Linn, and McCarthy. They agreed that it is preferable not to treat Class I cases at the age of 6 to 7 years. The majority felt that it is wise to treat 6- to 7-year-old Class II cases, but agreed that a second treatment should be planned. Drs. McCoy, Gray, Chuck, Nance, Reese and Oppenheim discussed the subject from the floor, the majority being in favor of early treatment. Dr. Oppenheim urged early treatment with light force applied only at night.

1946 MEETING

When the Office of Defense Transportation withdrew all restrictions on conventions after V-J Day, orthodontists up and down the Coast began to look forward to our regular February Meeting. However, the San Francisco Convention and Tourists Bureau immediately issued a statement "that the lifting of the ban would make no difference in this city until well into 1946." The rapid release of servicemen in the Pacific has created extreme congestion in the area. Nearly a million and a half men will arrive during the next seven months. It is stated that February, March, and April will be heaviest. Officers and officials have priority rating at all hotels, which are crowded to the utmost and will continue to be, during this migration.

Our committees have been studying the situation and have come to the conclusion that it is not wise to attempt a February, 1946, meeting.

Dental Corps Officer Honored by British

Captain Robert E. Moyers, Dental Corps, of Guthrie Center, Iowa, was made an Honorary Member of the Order of the British Empire by the Earl of Halifax at a special formal ceremony at the British Embassy on October 5. The citation follows:

"This officer parachuted into Roumeli, Greece, in January 1944 and from that time until the Germans left in October 1944 he served as the only medical officer in that part of Greece.

"He organized hospitals and relief and medical services in central Greece, an undertaking which involved him in considerable travel and hardship during the winter and early spring of 1944. In addition he always made himself and his staff available for operations whenever called upon. In particular he accompanied British troops in a large scale attack on Kaitza railway station on June 10, 1944 and took charge of Greek and mission casualties that occurred there with complete disregard for his own safety.

"A German drive into the area Lamia-Karpenisi took place in August 1944 and Captain Moyers took charge of the relief of the area. He visited burned and distressed villages while the German drive was still in progress, organized medical relief and field kitchens. At the end of September 1944 he had set up a system of relief on so efficient a basis that it has since been taken over complete by AML.

"The work of this officer proved of immeasurable benefit, not only to members of the Allied military mission but also to the resistance movement as a whole; the area in which he worked is recognized as having suffered more heavily than any other part of Greece from German reprisal.

"The good will of the people to the resistance movement until the final evacuation of the Germans was due in a large degree to the inspired and untiring work of Captain Moyers."

Army Dentists Fill Over Seventy-One Million Teeth

The Army Dental Corps has made 71,500,000 fillings, 16,500,000 extractions, and 2,600,000 dentures for personnel of the Army since Pearl Harbor, according to a report by Major General Robert H. Mills, Director of the Dental Division, Office of The Surgeon General.

As a result of the dental requirements of the early Selective Service program from 1940 to 1941, which required the recruit to have three natural masticating teeth and three serviceable opposing natural incisors, 8.8 per cent of the first 3,000,000 inductees were rejected. Dental defects were the leading cause for rejection, with eye defects second, and mental and nervous defects third. The dental requirements for entrance in the Army were lowered twice in 1942 to meet the need for men in military service and finally the only disqualifying factors were severe jaw malformation or malignancies.

The Army Dental Corps accepted full responsibility for rehabilitating these men who could not meet the minimum dental requirements. It was found that average dental requirements for every 100 inductees were about 60 to 80 extractions and a minimum of 240 fillings.

In addition to the rehabilitation of 1,500,000 men for the Army by use of prosthetic appliances, the Army Dental Corps is now working in cooperation with ophthalmological services in methods of fabricating an artificial eye in synthetic resin, of which more than five thousand have been inserted to date. The Dental Service has been active also in making and perfecting the technique associated with plastic ears, noses, and chins.

Notes of Interest

Morse R. Newcomb, D.D.S., announces his return to the practice of orthodontics, 964 Rose Building, Cleveland, 3494 Lee Road, Shaker Heights, Ohio.

William E. Koch, Jr., D.D.S., announces the opening of an office for the practice of dental radiography and diagnosis, 615 Metropolitan Building, St. Louis 3, Missouri. JE 9493.

Wilbur D. Johnston, Major, M. C., announces his return to the practice of orthodontics, 215 Whitney Avenue, New Haven, Connecticut. Telephone, 5-4211.

Major Robert E. Hennessy, Army Dental Corps, announces the reopening of his office at 929-934 University Club Building, Grand and Washington Avenues, St. Louis 3, Missouri. Practice limited to orthodontics. JE 0967.

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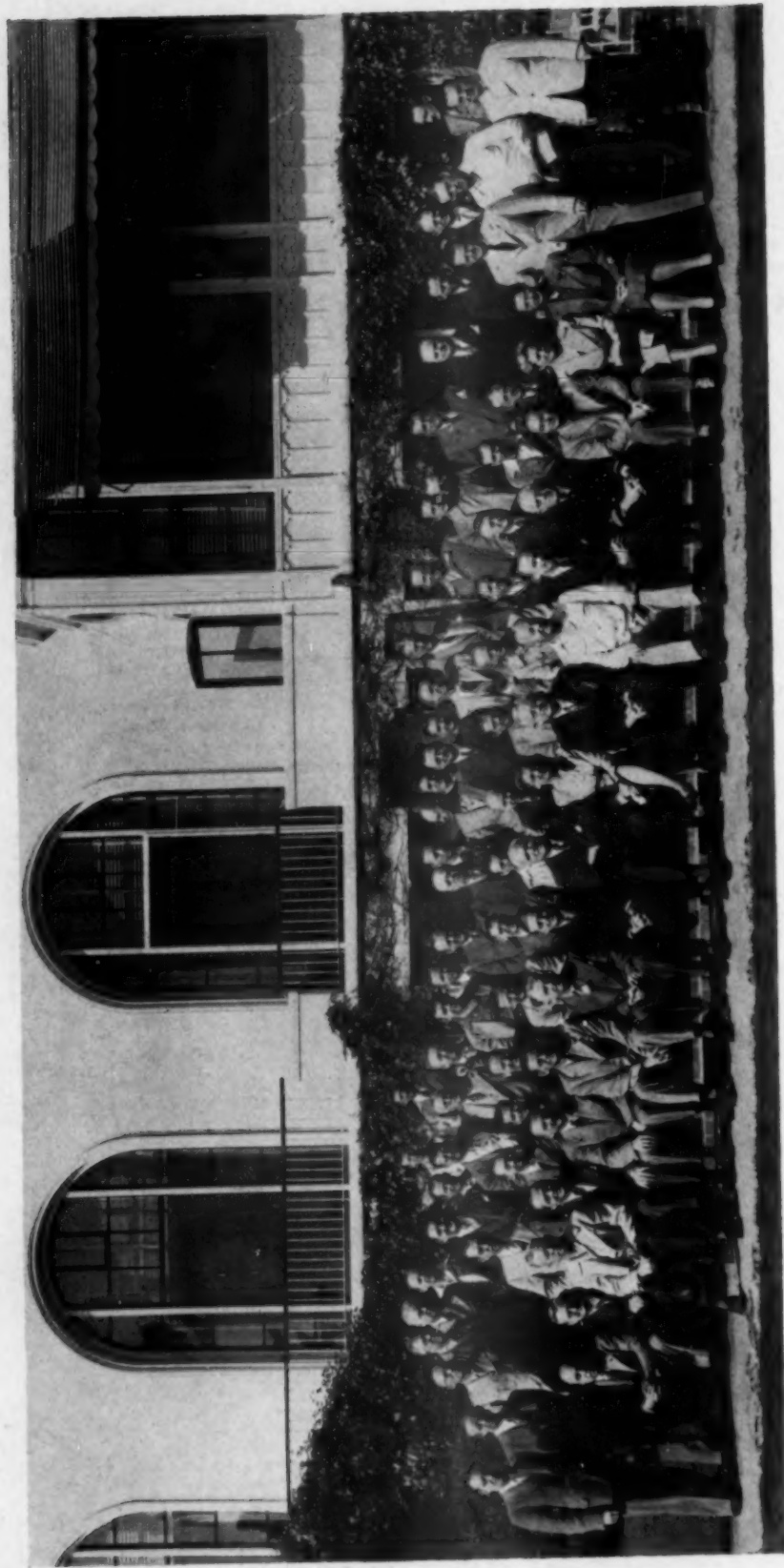
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FIRST DENTAL MEDICINE SEMINAR

Del Monte Lodge, California • October, 1944

University of California

Dental Medicine

Number

FROM THE DIVISION OF DENTAL MEDICINE, COLLEGE OF DENTISTRY, THE GEORGE WILLIAMS HOOPER FOUNDATION FOR MEDICAL RESEARCH, AND THE INSTITUTE OF EXPERIMENTAL BIOLOGY, UNIVERSITY OF CALIFORNIA

WITH SELECTED ARTICLES FROM THE FIRST ANNUAL SEMINAR FOR THE STUDY AND PRACTICE OF DENTAL MEDICINE

INTRODUCTION

IN DENTISTRY the practitioner finds himself possessed of a great tendency to ignore the notion that he treats living tissues. He finds his attention fixed on the problems of repair of the hard tissues of the teeth and their replacement by inert materials. Of course, from time to time an obstreperous case upsets his routine. On one occasion a patient has so many cavities he cannot fill them rapidly enough to keep abreast of the rampant decay and even the fillings he can place are lost because of secondary decay. It is easy enough to place full dentures and eliminate all these difficulties. Sometimes though, the dentist is shocked by this idea because of the age, appearance, or occupation of the patient. Then he wonders about the possibility of prevention. On another occasion it is the gingival tissues which do not respond to his ministrations to the hard structures. Removal of calculus, smoothing of rough margins, and grinding for overocclusion still leave the teeth loose, and the placement of dentures is considered a good solution. This time the problem is hardly met because the alveolar process does things that it should not. The dentures do not remain comfortable and relining is frequent.

The Division of Dental Medicine of the University of California presents these papers as evidence of the mounting interest in the biologic background of dentistry's problems. The staff members of the Division have been joined in these presentations by several lecturers from the First Annual Seminar for the Study and Practice of Dental Medicine, which took place at Del Monte Lodge, Pebble Beach, California, from October 15 to 19, 1944. These men represent the broad fields of psychosomatic medicine and dentistry, laboratory medicine, endocrine medicine, internal medicine, bacteriology, preventive medicine, biochemistry, neurology, and biometry.

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EDITORIAL

Dental medicine has progressed in the West from the establishment of the Division of Dental Medicine in the College of Dentistry of the University in California in 1934 to the development of periodic postgraduate courses in this field in various communities in northern and southern California as well as in the Northwest and several midwestern and southern states. Under the chairmanship of Hermann Becks, a staff of energetic workers has organized the dissemination of knowledge in the field of prevention and dentomedical relationships for organized dentistry and at the same time has contributed greatly through numerous investigations to our knowledge of the many biologic aspects of dentistry's problems. Experimental researches in biochemistry and pathology have been conducted by this Division in the fields of osteodystrophies, endocrinopathies, salivary chemistry, mineral and vitamin deficiencies and overdoses, etc. Clinical studies have been conducted on the systemic background of paradentopathies, caries immunity and susceptibility, dental health capacity, idiopathic root resorption, orthodontic prognosis, and many other problems. Critical analyses of the literature and biometrical evaluations have accompanied most of these studies. The Division has produced color motion pictures of clinical and surgical aspects of oral lesions, technical procedures for caries prevention, and various phases of roentgenographic interpretation in dentistry. Parallel with these investigations the staff of the Division has conducted an extensive program of practical caries prevention.

The opening of the First Annual Seminar for the Study and Practice of Dental Medicine marked a unique event in dentistry. Last October, eighty dentists of the West gathered at Pebble Beach, California, and found provided for them a carefully organized and punctual four-day program led by such outstanding men as Hermann Becks, Douglas Campbell, Jesse Carr, Herbert M. Evans, Arthur L. Jensen, Admiral C. H. Mack, Stacy R. Mettier, Karl F. Meyer, Michael J. Walsh, Robert Wartenberg, and Wendell L. Wylie. The purpose of such seminars is to bring together once a year interested professional men for a four-day period and to present the latest advances in the form of a concentrated course of instruction and demonstration, and to do this in an atmosphere also permitting rest and relaxation. It is generally felt that the progress of dentistry along the lines of prevention is possible only through a better understanding of the fundamentals of the basic sciences and biology as well as those of internal medicine and other allied and borderline fields of dentistry and medicine.

Many postgraduate educational courses have had the handicap of not allowing sufficient time and opportunity for free and open discussion of subjects presented in lectures and demonstrations. Since collectively much is gained by individual participation, the "seminar plan" is favored because it represents an attempt to learn through the combined efforts of the whole group as well as those of the clinicians and instructors. With the cooperation of all participating, this plan provides a most advantageous consideration of each subject with an unusual freedom from prejudice and dogma. Also, in order to emphasize the seminar character, participants are limited to those actually in residence at the place of meeting for its duration. This similarly applies to essayists and contributors who are invited contingent upon this policy. Thereby association and interchange are enhanced by participants and contributors living together, working together, and studying together for the entire interval of four days.

One of the most striking aspects of the development of dental medicine on the West Coast is the fact that even though the nucleus of this new movement originated with the staff of the Division of Dental Medicine of the University of California, the dental profession *at large* is recognizing the importance of this field for the progress of dentistry. Evidence of this trend is expressed by the recent sponsorship and approval of future Dental Medicine Seminars by most western State Dental Associations.

K. H. T.

COMPARATIVE ROENTGENOGRAPHIC AND HISTOLOGIC STUDY OF HUMAN MANDIBLES

HERMANN BECKS, M.D., D.D.S., AND DAVID H. GRIMM, D.D.S., M.S.,
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INTRODUCTION

THE inadequacy of our knowledge in the field of roentgenographic interpretation in dentistry has been generally recognized. Weski⁶⁸ and Euler²⁷ were among the first investigators to compare the roentgenographic and histologic aspects of human jaws. Their studies dealt with an exploration of periapical lesions and other diseases of the paradentium because of the frequent differences between the diagnosis of the roentgenologist and the actual microscopic findings. Since the roentgenographic appearance of tissues may easily be misleading, many phases of dental roentgenographic interpretation need to be explored. In comparing dental roentgenograms one is struck by the wide variations in the appearance of the bony architecture, and additional comparative roentgenographic and histologic studies may provide a better foundation for the interpretation of the fine distinctions between normal and pathologic conditions of the jawbone.

Investigations of the inner structure of bone date back to the fundamental observations of Gallilei (1638),³⁰ Duhamel (1760),²⁵ von Loder (1805),⁵⁷ Bour-gery (1831),²² Ward (1838),⁶⁵ Wyman (1857),⁷⁷ Engel (1851),²⁶ and Humphry (1858).³⁹ In 1867 the anatomist, Hermann von Meyer,⁵⁸ demonstrated in collaboration with Culmann, an outstanding Swiss mathematician, that the trabeculae of cancellous bones are arranged according to the various stresses within the bone. This discovery forms the basis of our present understanding of the functional arrangement of bone structure. The extensive work that followed these earlier investigations of the functional arrangements of the spongiosa finally led to the concept of "trajectors."⁵⁹⁻⁶¹ The discovery of roent-gen rays resulted in a considerable increase of knowledge about these trajectorial systems, particularly the changes under pathologic conditions. The *pars spongiosa* was thought of as having a special functional importance while the *pars corticalis* was explained as a firm hull in which the spongiosa is anchored. In 1869 Wolff⁷⁰ demonstrated in very thin sections the detailed structure of bone and published in 1892 his classic contributions on the law of bone transformation.⁷³ This law was based on the earlier mathematical analysis of Culmann and Wolff's observation that the inner architecture of bone, normal or pathologic, is governed by the static conditions.^{71, 72, 74, 75} Many controversies developed over the doctrines of Wolff (Zschokke,⁷⁸ Lorenz,⁴⁴ Bähr,³⁻⁵ Ghil-lini,^{32, 33} Schede⁵²), but in general the fundamental observations by Culmann and Wolff were confirmed (Albert,¹ Friedlander,²⁸ Fuld,²⁹ Gebhardt,³¹ Koch,⁴² Maas,⁴⁵ Roux,^{48, 49} Schmidt,⁵³ Sudeck,^{55, 56} von Recklinghausen,⁶² Weichsel-baum,⁶⁷ and others).

In 1921 Winkler⁶⁹ observed the increased thickness of the *pars compacta* in the region of the diaphysis of long bones, which led to his examination of the

From the George Williams Hooper Foundation for Medical Research and the Divisions of Dental Medicine and Dental Surgery, College of Dentistry, University of California, San Francisco, California.

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osseous structure of the jaw of a microcephalic Cebus monkey to determine whether or not the *pars compacta* has a special functional importance. Labiolingual and longitudinal sections were prepared, both by decalcifying and grinding techniques. He demonstrated the various trajectorial systems and the distribution of compacta and spongiosa of the jaws. The procedure of Winkler offers new possibilities for the study of human jaws under normal as well as pathologic conditions. By means of comparative roentgenographic and histologic studies it is possible to advance our knowledge of the inner morphologic detail of jaws, thus enabling the dentist to interpret roentgenograms more accurately and increase their value in dental practice.

Roentgenographic Considerations.—Three techniques are available for the study of the spongy structure of the mandible. The oldest one is the preparation of ground sections. This method, however, has certain disadvantages. Because of frequent interference by the teeth, there is difficulty in obtaining sections of the same cutting direction. The spongiosa is often damaged during the grinding, and fine trabeculae may be torn loose and washed out during the process. Organic remnants may simulate structural conditions which in reality do not exist. Finally, large parts of the specimen are destroyed by cutting out the various blocks for preparation.

A second method is the decalcification of the specimen and the preparation of exceedingly thin serial sections in longitudinal and buccolingual directions. This procedure without doubt gives the most information but requires much time.

Roentgenography serves admirably as the third method. The first roentgenograms for the determination of detailed osseous structure were made in 1898 by O. Walkhoff⁶³; he produced them for a paper on vertebrae which was presented by Beneke.²¹ At the same time Walkhoff called attention to the great value of roentgen rays as a means of diagnosing changes in the osseous structures of the face. Soon after, Gocht²⁴ and Hoffa³⁸ described structural features of long bones with the aid of roentgen rays. In 1900 Wolff⁷⁶ prepared a large series of roentgenograms demonstrating the internal architecture of bones. In 1902, Walkhoff⁶⁴ published an extensive report on the functional development and form of the mandible in anthropomorphs and man, based on the use of roentgen rays. In 1911 Ruediger⁵⁰ differentiated bone structure roentgenographically into *pars corticalis*, *spongiosa*, and *medullaris*. Dieck,²⁴ in 1911, called attention to differences in the density of bone structure under pathologic conditions. Normal atrophy of bone structure in senility by the uniform rarefaction of bone trabeculae was described by Grashey³⁵ in 1913 in his textbook of roentgenographic diagnosis. The reports in 1918 by Braunschweiger²³ and in 1921 by Winkler⁶⁹ contributed considerably to our knowledge of the distribution of bone trabeculae in the mandible.

Clinicians have been greatly handicapped in their interpretation of the osseous structures shown in dental roentgenograms because of lack of uniformity in technical procedure, especially with respect to angulation, exposure, and development. In most instances, roentgenologists, dentists, or technicians concentrate all their efforts on producing roentgenograms which demonstrate coronal or periapical structures without giving much attention to the correctness of tooth form or interdental bone structure.

It is obvious that if the wrong angulation leads to a distortion, i.e., an elongation or shortening of the image of a tooth, it will also produce distorted

views of the surrounding bone structure which would be inadequate for interpretation of morphologic changes. By this means the alveolar crest frequently appears to have been resorbed, simulating vertical or horizontal atrophy; the alveolar bone or lamina dura cannot be followed around the tooth. Overexposure and overdevelopment frequently are evident as a partial burning out of the alveolar crest. Thus, not having a detailed picture, it is sometimes impossible to note pathologic changes such as abnormalities of the vascular system within the bone, or osteodystrophic lesions.

It must be emphasized, therefore, that the accurate interpretation of dental roentgenograms depends upon the highest standards of roentgenographic technique. With accurate technique it has been possible to recognize, roentgenographically, changes in the alveolar process indicating generalized metabolic disturbances which had not been disclosed by physical examination, or had not yet produced clinical manifestations. It has been possible to determine that progressive alveolar bone changes were the result of factors not related to dental disturbance whatsoever. These possibilities emphasize the need for the establishment of normal standards of bone structure. Such standards are also necessary in the field of parodontics because of the importance of the early recognition of primary bone changes which are frequently the initial stage of certain types of parodontopathies.^{15, 66}

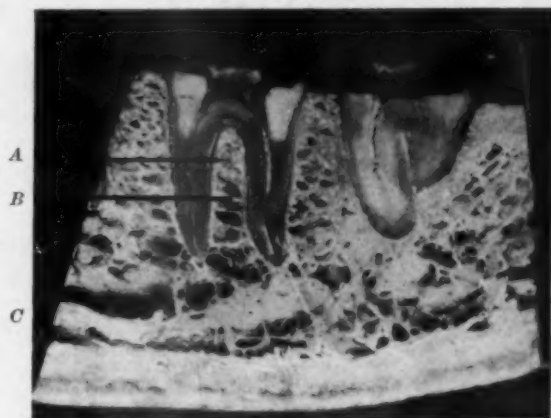


Fig. 1.

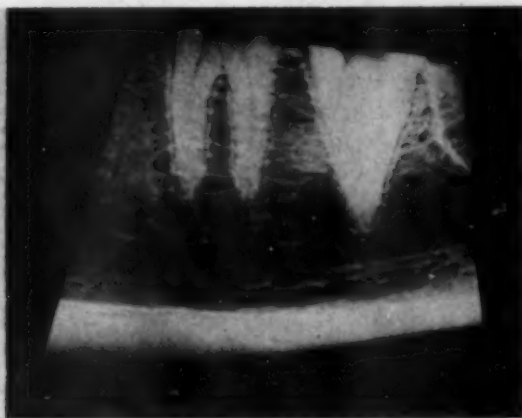


Fig. 2.

Fig. 1.—Dry specimen from human mandible demonstrating distribution of bone structure in the region of the lower first and second molars (natural size). A, Alveolar bone; B, supporting bone (trabeculae); C, mandibular canal. (Pl. 726.)

Fig. 2.—Roentgenogram of specimen in Fig. 1 (Pl. 1032).

From the standpoint of roentgenographic interpretation the bone structure of the jaws may be divided into alveolar bone (lamina dura) and supporting bone (trabeculae). This classification has proved most practical because the effects of exogenous and endogenous factors will not always involve both of these types of bone structure at the same time. It is well known that osseous tissue will undergo disuse atrophy if it does not receive functional stimulation. If such functional stimulation is not received by both the alveolar bone and supporting bone, definite changes will occur which will express themselves first in the disappearance of supporting structure. In other words, in cases of disuse atrophy due to loss of opposing teeth, definite signs of resorption of the fine meshwork of bone trabeculae are found. Other possible biologic significance of the presence or absence of supporting bone has been shown by Aub,² Becks,^{6, 7, 9, 11, 12, 13, 14, 15} and others.

The distribution of bone structure in the paradentium of a lower first molar is demonstrated in Figs. 1 and 2. Fig. 2 shows the roentgenographic appearance of the dry specimen of Fig. 1. The root surfaces are surrounded by a distinct plate of alveolar bone and between these, under the bifurcation and in the interdental space between the distal root of the first molar and mesial root of the second molar, are crossbars which *support* the alveolar bone. These crossbars lend functional "support" to the alveolar bone proper. The denser the supporting meshwork of bone structure, the firmer is the fixation of the tooth in its alveolus. The more distinct the appearance of trabecular structure as fine radiopaque lines over the apical third of roots, the denser is their structure, which is an important prognostic criterion for surgical procedures.

The adaptation of the osseous structure to changed functional requirements is demonstrated in serial roentgenograms following the extraction of teeth (Fig. 3).*

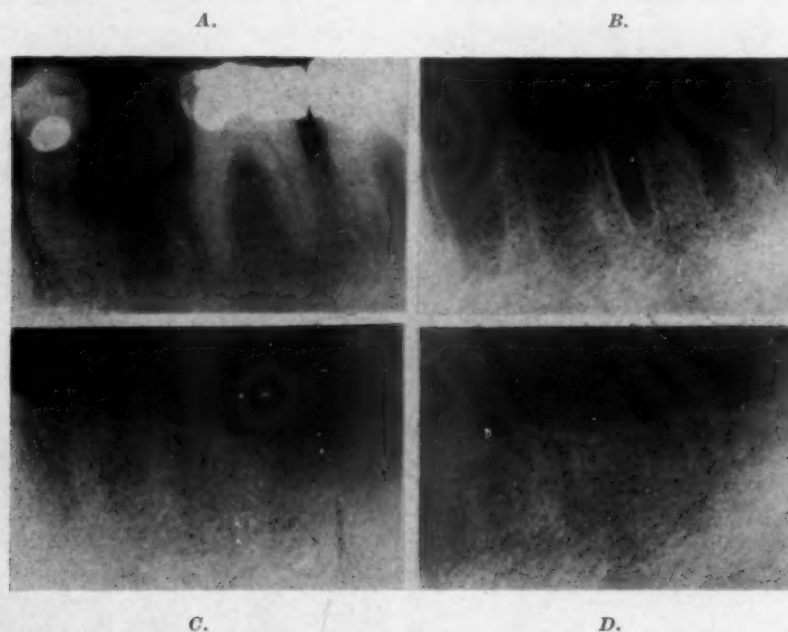


Fig. 3.—Roentgenograms before and after the extraction of lower left first premolar and first and second molars. *A*, Before extraction; *B*, One month after extraction; *C*, eighteen months after extraction; *D*, twenty-four months after extraction. Note remaining alveolar bone in *B* and absence in *C* and *D* with reorientation of supporting bone. (Pl. 1834, 1833, 1832, 1831.)

The roentgenogram of Fig. 3, *A* shows the lower left first premolar and first and second molars present in the jaw of an individual 45 years of age. The second premolar had been removed several months previously. The roentgenogram of Fig. 3, *B* shows this area one month after the extraction of the first premolar and first and second molars. Note the distinct outline of the alveolar bone, appearing as vertical radiopaque lines, the radiolucency of the socket, and the regular distribution of bone trabeculae between the plates of alveolar bone. Eighteen months later (Fig. 3, *C*) the vertical opaque lines have lost their distinct appearance and the entire supporting bone structure appears to be undergoing reorganization. Twenty-four months later (Fig. 3, *D*) the reorganization is almost complete. Note the changed architecture of the jawbone structure which has adjusted itself to new functional requirements. Such changes in the paradental osseous structures are in conformity with observations in other bones of the skeleton following changed functional stresses.

*Through the courtesy of Dr. Frank Williams, Pasadena.

Experimental Observations.—In the course of recent experimental studies in this laboratory, it was observed that the structure of the jawbone could be altered in animals by nutritional means.^{10, 15} In other experiments, endocrinopathies such as lack of hormones of the anterior lobe of the pituitary gland or injections of adrenocorticotrophic hormone produced changes in the skeletal structure of long bones and jaws which were primarily of an atrophic or osteoporotic nature.^{16, 17, 18, 19, 20, 40, 41, 46, 47, 54} The gross and histologic studies of this material emphasize the fact that it is possible to influence the growth and development of bone structure in young and old dogs and rats at will by deficient diets and experimental endocrinopathies.

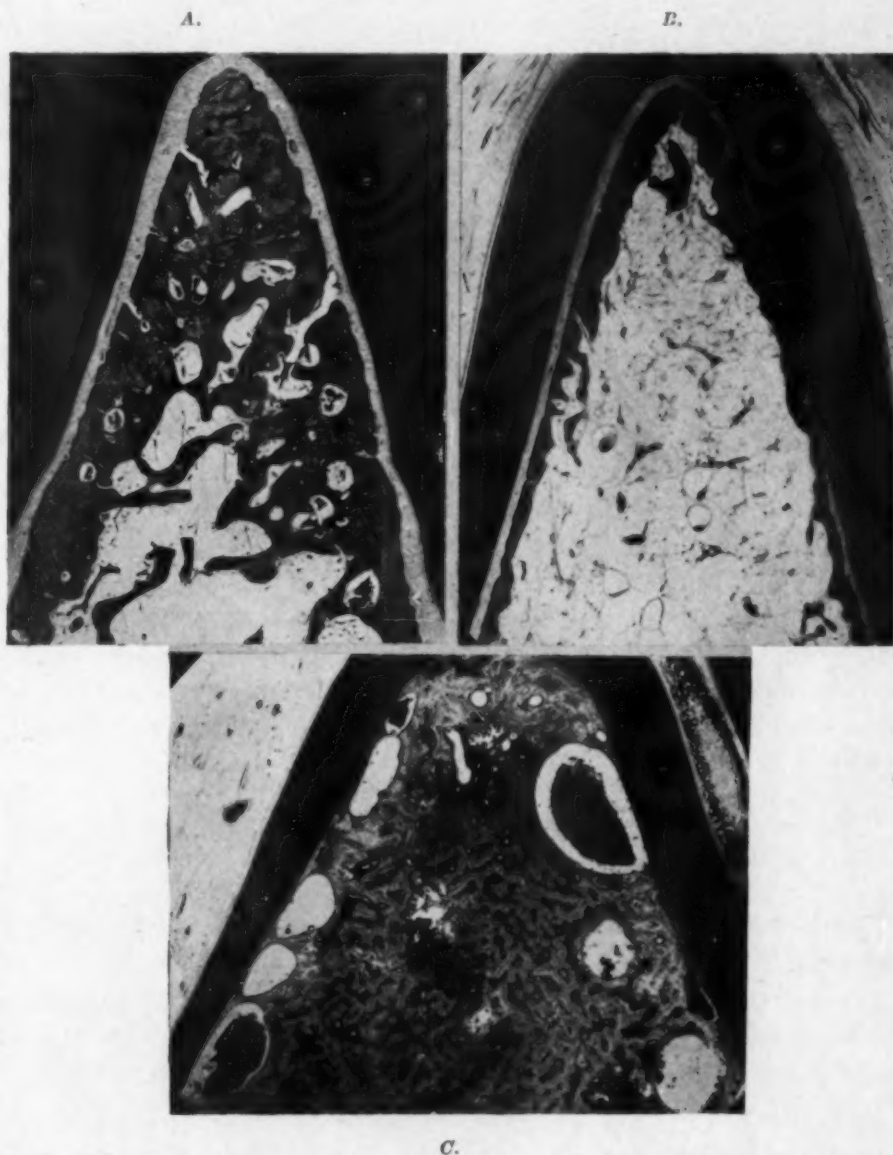


Fig. 4.—Changes in the paradental bone structure under the bifurcation of premolars of dogs as a result of dietary deficiencies. *A*, Normal distribution of alveolar and supporting bone with small marrow cavities; *B*, progressive bone atrophy (osteoporosis). The alveolar bone has been resorbed to a thin plate. Marrow tissue consists of fat; *C*, osteodystrophy with the formation of pseudo-cysts partially filled with blood. Newly formed bone structure consists of fibro-osteoid. (Pl. 229, 430, 527.)

Fig. 4 may serve as an example of such osseous changes taken from previous experiments^{7, 13, 15} on dogs. The distribution of alveolar bone and supporting bone under the bifurcation of a premolar of a normal control dog is seen in

Fig. 4, A. In Fig. 4, B the supporting bone has disappeared and is replaced by fat marrow in an experimental animal of the same age which had been on a vitamin D deficient diet for eight months. Note the conspicuous absence of bone trabeculae with only the alveolar bone remaining. This is the typical picture of *osteoporosis*. A similar condition was observed by Kronfeld in a human jaw as a result of disuse.⁴³ Fig. 4, C shows an extremely fine mesh of fibro-osteoid tissue with blood-filled marrow cavities and pseudo-cysts as they are found in von Recklinghausen's disease. This slide represents a typical osteodystrophy as a result of a deficiency of calcium and vitamin D in dogs; the lesion resembles the changes produced in animals and human beings by hyperparathyroidism.

The finding of these rather advanced changes in animals suggests that similar, or moderated forms may occur in human beings with a characteristic appearance in dental roentgenograms. In the following preliminary study an attempt has been made to correlate roentgenographic and morphologic changes in human jaws.

MATERIAL AND METHOD OF STUDY

Since it was difficult to obtain complete or even fragmentary human jaws sufficiently well fixed for histologic preparations, not to speak of material from representative age groups, it was necessary to utilize jaws of cadavers available for dissecting and instruction purposes. Fixation in some instances was only fair, and age data were lacking. However, since the purpose of this investigation was to compare the roentgenograms of these jaws with the morphologic appearance of bone tissue in labiolingual and buccolingual cross sections, the jaws of such cadavers offered suitable and readily available material.

The first step in the preparation of the material was to remove the soft tissue and to cut nicks in the lower border of the mandible to identify those areas which were later to be examined in stained sections. The jaws were then roentgenographed and cut into thin slabs with a nick in the center of each. Eleven specimens were thus prepared, decalcified in 5 per cent nitric acid in water, embedded in nitrocellulose, sectioned at 8 to 10 μ , and stained with hematoxylin and eosin.

ROENTGENOGRAPHIC AND HISTOLOGIC STUDY OF FIVE HUMAN JAWS

Jaw I

This left jaw fragment (Fig. 5) extended from the central incisor to the second molar region with $\sqrt{123456}$ present.

The jaw was cut into four portions (Specimens 1, 2, 3, 4) as indicated by the vertical lines (Fig. 5). The photomicrographs, Figs. 6, 7, 9, and 11, are taken from sections of these four specimens in the areas of the nicks.

SPECIMEN 1.—

Roentgenographic Aspect.—The roentgenogram (Fig. 6) shows a densely ossified area around the lower left cuspid. Note the finely dispersed bone trabeculae in the periapical regions about the lateral incisor, cuspid, and first premolar, and in the interdental spaces. The cortical plate appears very thick; the pulp canal of the cuspid is obliterated; the incisal surfaces indicate considerable attrition; and the peridental membrane spaces appear as thin radiolucent lines. Bone trabeculae appear as exceptionally fine radiopaque striae over the apical third of the roots indicating a dense and hard supporting struc-

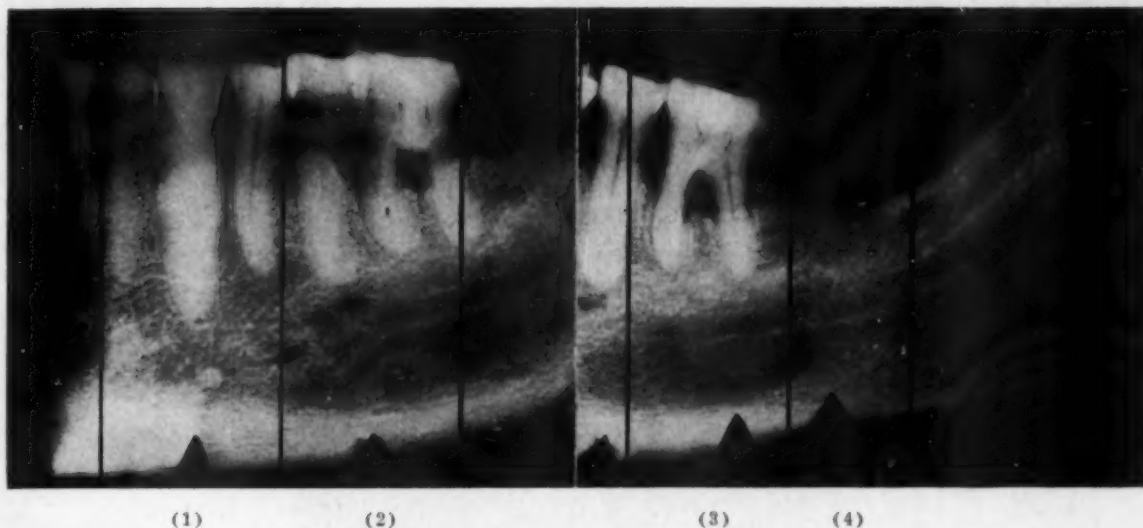


Fig. 5.—Jaw I. Roentgenographic reproduction. Jaw was cut into four specimens as indicated by the vertical lines (Specimens 1, 2, 3, 4). Histologic sections were taken from the areas of the nicks. (Pl. 5161, 5162.)

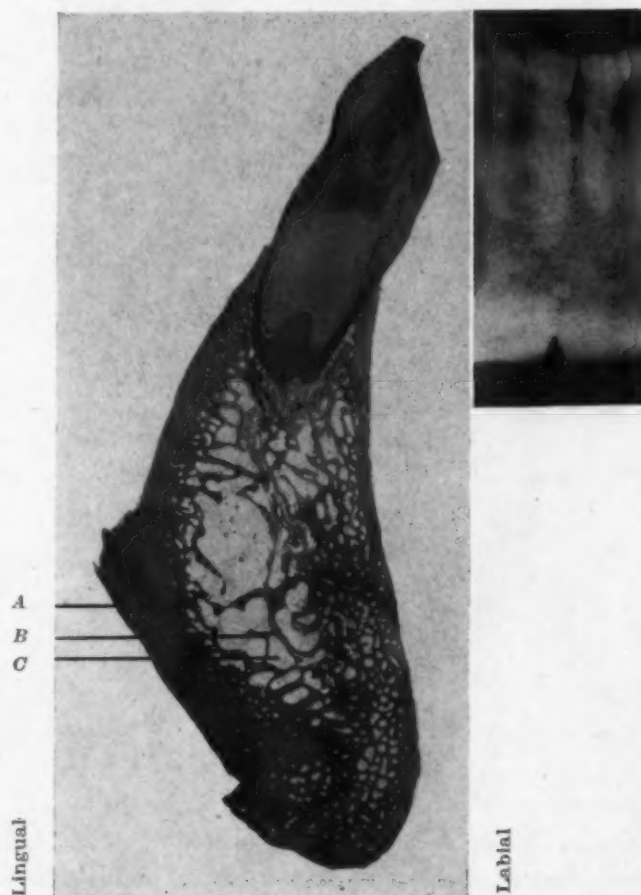


Fig. 6.—Jaw I. Specimen 1. Labiolingual section through "∧" in roentgenogram. A, Corticalis; B, bone trabeculae; C, marrow cavities. (Microsummar 80; distance from object 3 in.; extension of camera 16 in.; $\times 3.5$.) (Pl. 2047, 5161.)

ture. The alveolar crest is sharply set off against the background and the entire bone structure has a general slight radiopacity.

Histologic Aspect.—The cortical plate stands out as a particularly heavy layer of bone on the lingual side (Fig. 6, A) with only few marrow cavities; this density apparently accounts for the general radiopaque appearance over the entire roentgenogram. On the labial side and in the region of the lower border of the mandible the cortical plate is less dense and has a large number of small marrow cavities. Bone trabeculae cross from the labial to the lingual cortical plate and the marrow cavities are filled with fat tissue. The lower border of the mandible is especially dense and consists of well-organized shell bone or Haversian system bone. The soft tissue had been previously removed. The section through the tooth is slightly tangential.

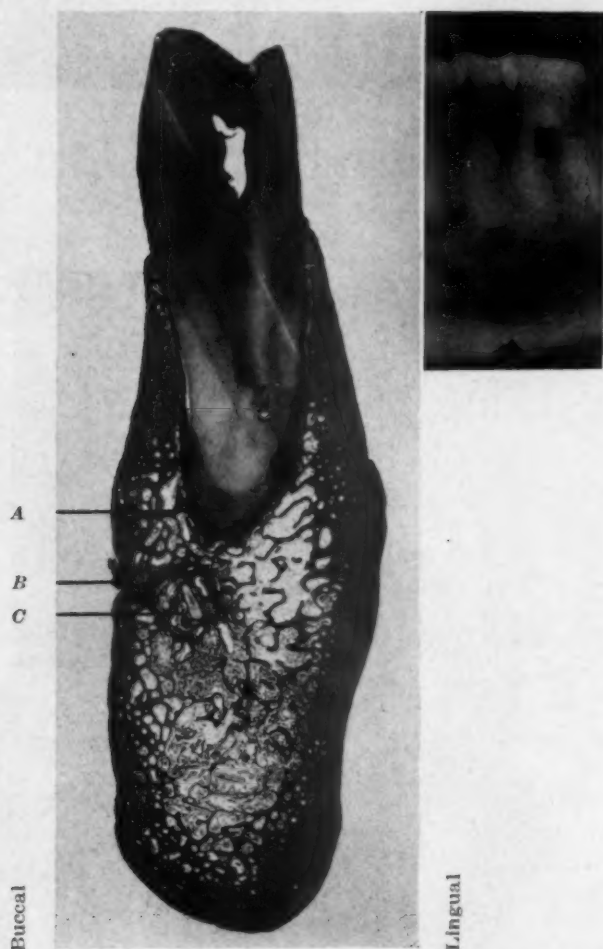


Fig. 7.—Jaw I. Specimen 2. Labiolingual section through “^” in the region of the second premolar. A, Hypercementosis; B, mental foramen; C, mandibular canal. (Microsummar 80; distance from object 3 in.; extension of camera 16 in.; $\times 3.5$.) (Pl. 1084, 5173.)

SPECIMEN 2.—

Roentgenographic Aspect.—This specimen (Fig. 7) is taken from the mandible of Jaw I (Fig. 5) in the area of the second premolar and through the mental foramen. The incisal surface shows considerable wear and although there is severe horizontal and vertical atrophy of the marginal bone structure, the distribution of bone trabeculae appears very dense. The mental foramen is clearly visible, and as in the former specimen the cortical plate of the lower border of the mandible is heavy.

Histologic Aspect.—As suggested by the roentgenogram the paradental bone structure (Fig. 7) is extremely dense. Only a few small marrow cavities can be noted in the immediate environment of the premolar root although they increase in size below the apex. The buccal and lingual cortical plates are approximately of the same width. The marrow cavities are filled with fat and some lymphatic tissue as seen in higher magnification of the periapical region in Fig. 8. The apical end of the tooth shows hypercementosis and the mandibular canal with its contents is seen in cross section opening to the buccal side. The histologic specimen confirms in detail the roentgenographic interpretation.



Fig. 8.—Enlargement of periapical area of Fig. 7. (Microsummar 42; distance from object 1 in.; extension of camera 22 in.; $\times 10$.) (Pl. 2037.)

SPECIMEN 3.—

Roentgenographic Aspect.—In the region of the first molar considerable vertical and horizontal atrophy of the paradental bone structure is seen (Fig. 9) with an involvement of the bifurcation. Otherwise the bone appears very dense in the immediate environment of the roots. The mandibular canal is clearly visible.

Histologic Aspect.—Fig. 9 shows the histologic aspect of a section through the distal root of the first molar. The bone structure appears just as dense as in the specimen of Fig. 7. The buccal and lingual cortical plates are heavy. The bone trabeculae are closer together and seen in nearly longitudinal direction toward the apex of the tooth, indicating their functional importance inasmuch as approximately half of the supporting bone structure has been lost as a re-

sult of marginal atrophy. There are also many bone trabeculae running buccolingually. The marrow cavities have a large amount of lymphatic tissue. As an accidental finding, a fracture of the cementum may be noticed on the buccal side. A magnification of the mandibular canal area with blood vessels and nerve tissue is given in Fig. 10.

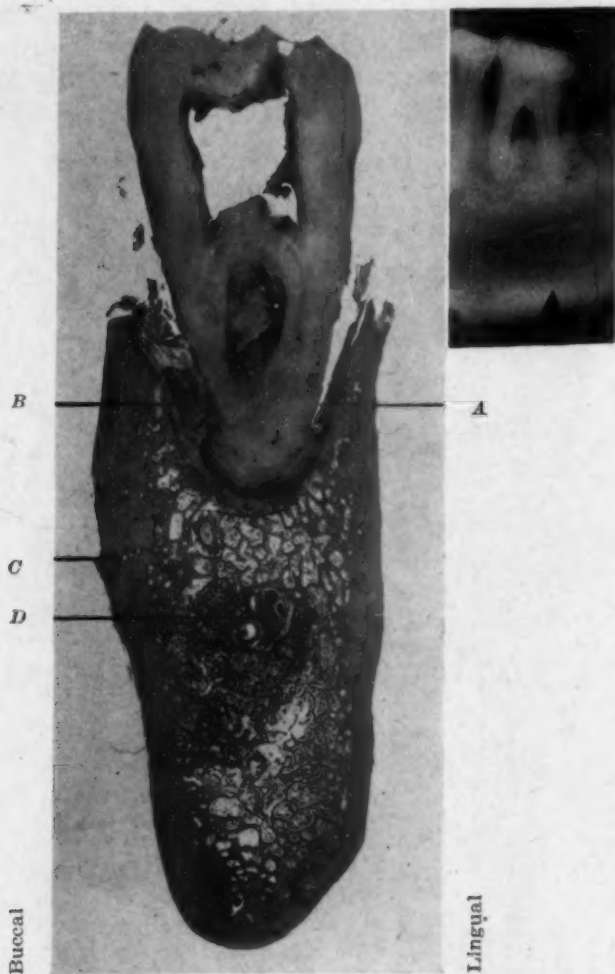


Fig. 9.—Jaw I. Specimen 3. Buccolingual section through “^” in roentgenogram. A, Artefact; B, fracture of cementum; C, corticalis; D, mandibular canal. (Microsummar 50; distance from object 3 in.; extension of camera 16 in.; $\times 3.5$.) (Pl. 1097, 5162.)

SPECIMEN 4.—

Roentgenographic Aspect.—This edentulous area as seen in Fig. 11 appears to be somewhat different from the foregoing specimen inasmuch as the distribution of bone trabeculae is less pronounced. The outline of the alveolar ridge on the distal side of the first molar is distinct; however, it does not show as sharp a radiopaque demarcation line as does, for instance, the lower border of the mandible. The mandibular canal is clearly visible.

Histologic Aspect.—It is interesting to observe that in this specimen (Fig. 11) the buccal and lingual cortical plates, especially above the mandibular canal, are considerably thinner than in the former specimen in which teeth were present. The arrangement of the bone trabeculae is irregular but dense. Some larger marrow cavities above and below the mandibular canal can be noted. Resorptive activity can be seen in several areas and the cortical plate is not closed in the ridge area even though some regeneration has taken place following the extraction of the second molar.

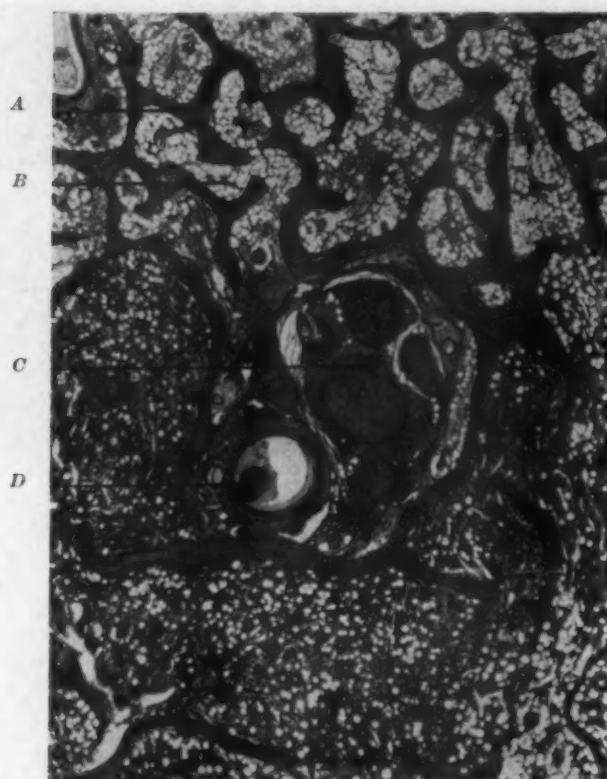


Fig. 10.—Enlargement of mandibular canal area of Fig. 9. *A*, Bone trabeculae; *B*, marrow cavities; *C*, inferior alveolar nerve; *D*, blood vessel. (Microsummar 42; distance from object 1 in.; extension of camera 28 in.; $\times 16.5$.) (Pl. 2038.)

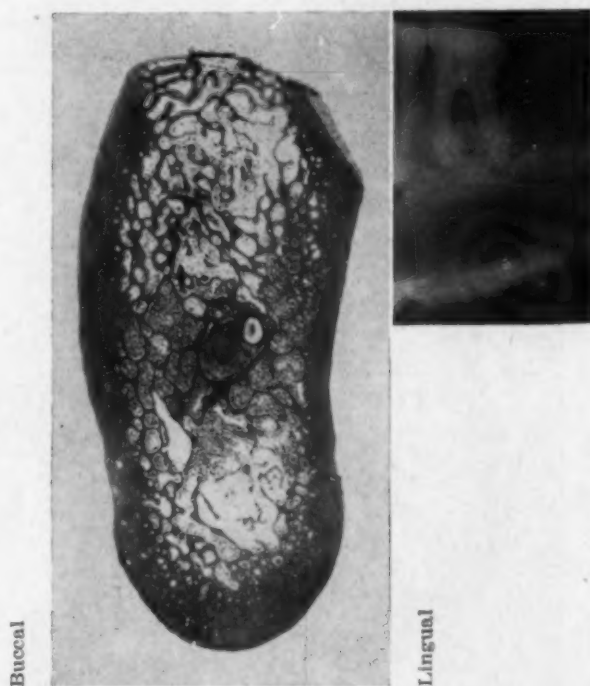


Fig. 11.—Jaw I. Specimen 4. Buccolingual section through " \wedge " in roentgenogram. (Microsummar 80; distance from object 3 in.; extension of camera 16 in.; $\times 3.5$.) (Pl. 5927, 5162.)



Fig. 12.—Jaw II. Roentgenographic reproduction. Two specimens were taken from the lower left cuspid and premolar areas (Specimen 5) and first molar area (Specimen 6) as indicated by the vertical lines.



Fig. 13.—Jaw II. Specimen 5. Buccolingual section through " \wedge " in roentgenogram. (Microsummar 80; distance from object 3 in.; extension of camera 16 in.; $\times 3.5$.) (Pl. 5928, 5163.)

Jaw II

This left portion of the mandible (Fig. 12) extended from the lateral incisor to the third molar area with all teeth present except the first molar. Two specimens were obtained, one from the lower left cuspid and premolar areas, and the other from the second molar area, as indicated by the nicks in this figure.

SPECIMEN 5.—

Roentgenographic Aspect.—The bony architecture of this specimen (Fig. 13) differs greatly from that of Jaw I. The trabeculae appear as a distinct radiopaque network and the marrow cavities are large. The lower border of the mandible consists of a heavy cortical plate. The mental foramen is located below the apices of the first and second premolars. There is considerable horizontal and vertical atrophy of the marginal bone structure. Contacts between cuspid and first and second premolars have been lost, apparently due to the extraction of the first molar. A slight radiolucency appears over the center of the root of the first premolar; the pulp canals are distinct.

Histologic Aspect.—A section through the area of the first premolar (Fig. 13) reveals densely ossified buccal and lingual cortical plates. On the lower border of the mandible the cortical plate has reached twice the width of the buccal and lingual plates. The section is slightly tangential which accounts for

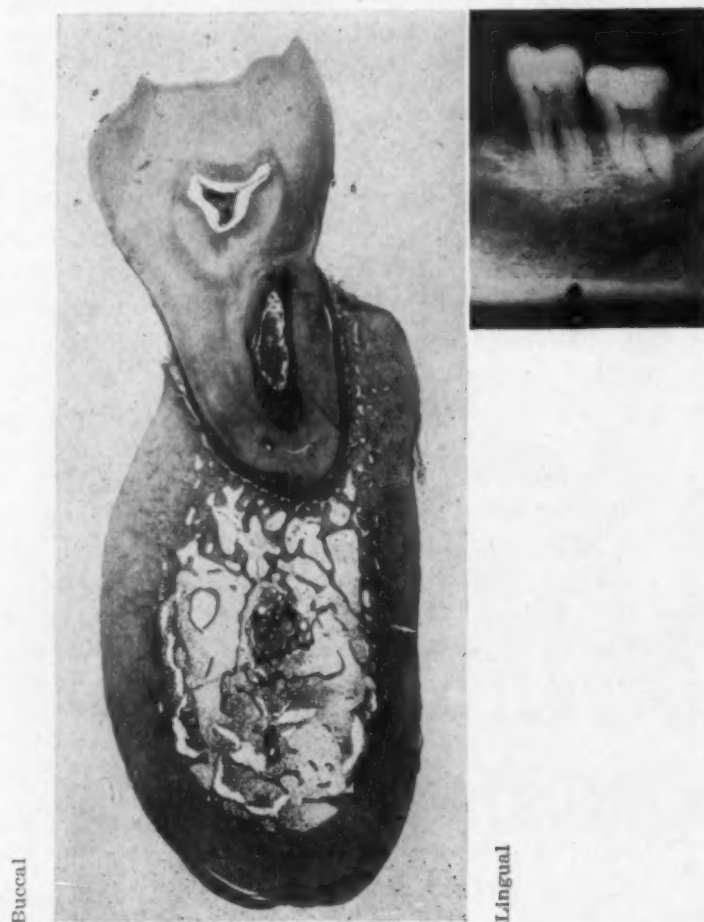


Fig. 14.—Jaw II. Specimen 6. Buccolingual section through “^” in roentgenogram (Fig. 12). (Microsummar 80; distance from object 3 in.; extension of camera 16 in.; $\times 3.5$.) (Pl. 1093, 5160.)

the absence of a cortical plate on the buccal side in this photomicrograph. In other sections of this specimen, not suitable for photomicrography, the paradental bone structure on the buccal side reached as high as on the lingual side. Quite in contrast to Jaw I large marrow cavities can be noted, bone trabeculae in the buccolingual direction are sparse; only in some instances do they connect transversely. The marrow cavities are filled with fat tissue. The central portion of the pulp canal is visible and a large portion of the dentine has been replaced by osseous structure following an extensive resorptive process which has reached the surface of the cementum, buccally as well as lingually. This pathologic process accounts for the oval-shaped radiolucency seen in the accompanying roentgenogram.

SPECIMEN 6.—

Roentgenographic Aspect.—The osseous structure in the apical region of the second molar in this specimen is dense (Fig. 14) and horizontal atrophy has involved the bifurcation. The mandibular canal is visible and the lower border of the mandible is half as thick as in the premolar area (Fig. 13).

Histologic Aspect.—The cortical plate is of even width and appears densely ossified (Fig. 14). The distribution of bone trabeculae near the apex appears denser than in the lower portion of the jaw between the mandibular canal and lower border of the mandible. The pattern of the supporting bone is entirely different from that in the premolar area and there is considerable lymphatic tissue in the marrow cavities. Again the histologic aspect confirms in detail the roentgenographic analysis.

Jaw III

SPECIMEN 7.—

Roentgenographic Aspect.—This jaw (Fig. 15) consisted only of a small part of the lower left mandible with $\sqrt{3456}$ and a root remnant of $\sqrt{7}$ present. All teeth showed considerable wear. The bone structure in the premolar area from which histologic sections were obtained shows an irregular pattern with a large radiolucent area below the mental foramen. There is some horizontal atrophy of the marginal bone structure and extensive hypercementosis.

Histologic Aspect.—The histologic specimen from the area over the nick is given in Fig. 15, and passes through the mental foramen. In comparison with the former specimen the cortical plate appears much thinner and the supporting bone around the surface of the tooth shows no specific orientation. Considerable resorptive activity can be noted in this specimen, and apparently as a result of it, all bone trabeculae have disappeared between the mental foramen and the cortical plate of the lower border of the mandible which accounts for the radiolucency in the accompanying roentgenogram. The alveolar bone bordering the periodontal membrane fibers has reduced to a fine line and the deposition of a heavy layer of secondary cementum can be noticed.

Jaw IV

This right mandible was not covered with soft tissue at the time it was obtained for this study. It was divided into Specimens 8 and 9 (Fig. 16).

SPECIMEN 8.—

Roentgenographic Aspect.—This specimen (Fig. 17) gained special interest because of the radiolucent vertical lines which run from the center of the

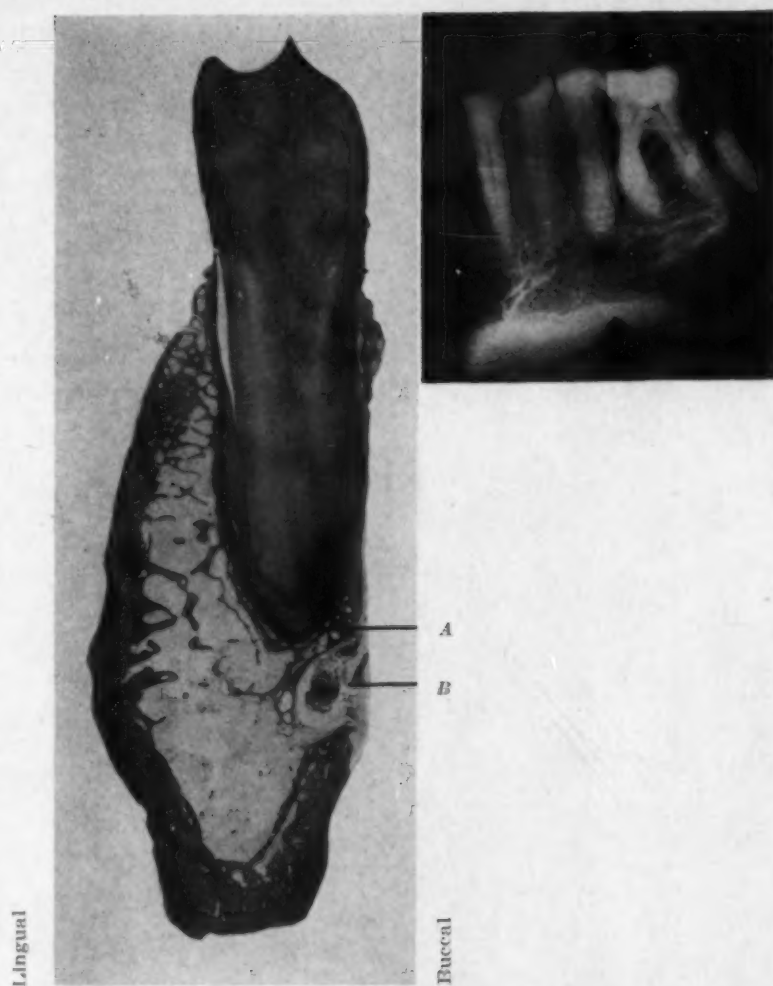


Fig. 15.—Jaw III. Specimen 7. Buccolingual section through " \wedge " in roentgenogram. A, Hypercementosis; B, mental foramen. (Microsummar 80; distance from object 3 in.; extension of camera 16 in.; $\times 3.5$.) (Pl. 2044, 1916.)

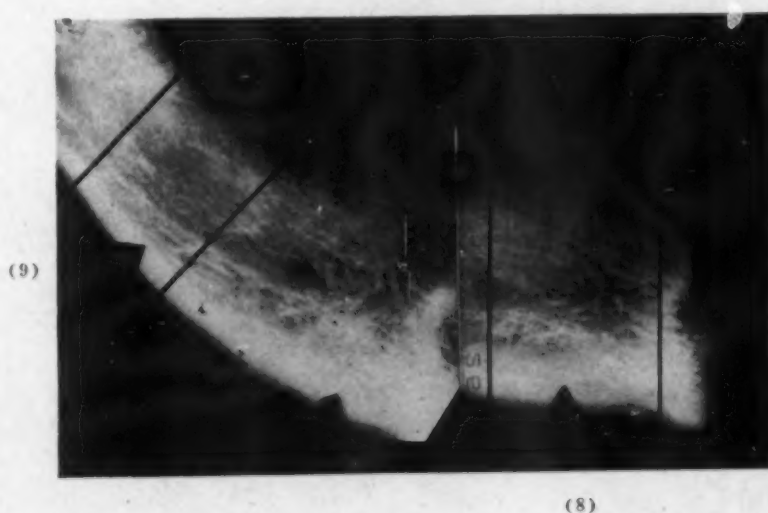


Fig. 16.—Jaw IV. Roentgenographic reproduction. The edentulous jaw was divided into Specimens 8 and 9 as indicated by the vertical lines. (Pl. 1919.)

mandible in the cuspid and premolar regions toward the alveolar ridge. They differ in width but are approximately parallel. Near the lower border of the mandible ossification is especially dense.

Histologic Aspect.—Fig. 17 is a reproduction of a slide through the nick of the accompanying roentgenogram and represents a section through such a radiolucent vertical line. It can be noted that a blood vessel runs from the center of the upper half of the mandible through the labial cortical plate. The cortical plate around the canal is dense and thick. Bone trabeculae are seen as heavy bars crossing labiolingually. The lingual cortical plate near the alveolar ridge is especially heavy with a few isolated marrow cavities; it has joined the labial cortical plate to be separated in this section only by a blood vessel.

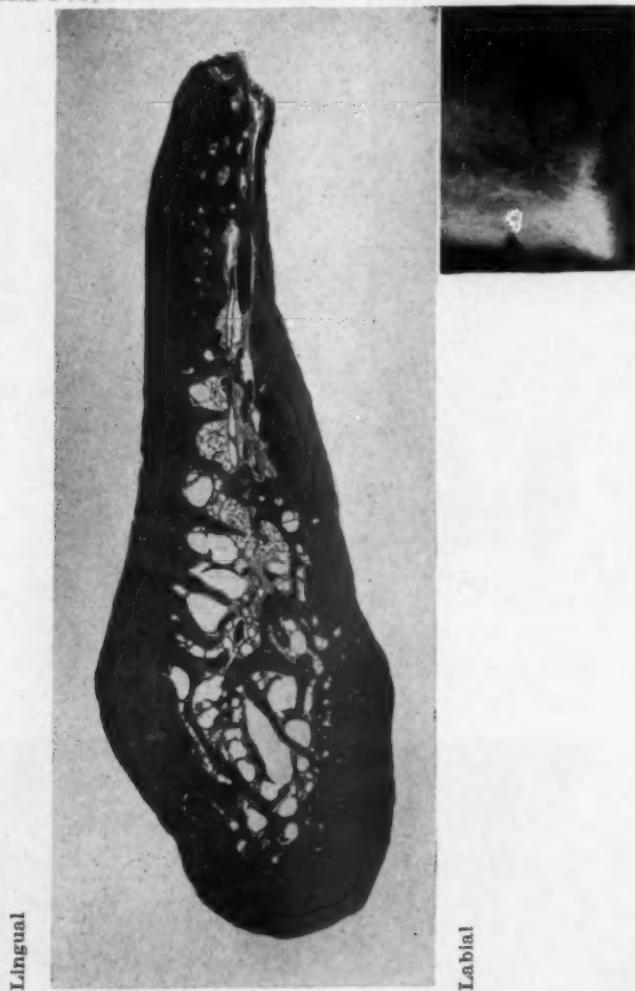


Fig. 17.—Jaw IV. Specimen 8. Labiolingual section through “^” in roentgenogram. (Microsummar 80; distance from object 3 in.; extension of camera 16 in.; $\times 3.5$.) (Pl. 1096.)

These bony channels originally described by Zuekerkandl^{79, 80} and further investigated by Hirschfeld^{36, 37} may in some instances have important diagnostic value as shown by Ryder.⁵¹ A magnification of the upper half of this mandible is given in Fig. 18. It is interesting to observe that the alveolar ridge at *B* does not consist of solid bone structure but that it has an opening due to this vessel-carrying bone canal.

SPECIMEN 9.—

Roentgenographic Aspect.—This specimen from the first molar area presents a uniform distribution of bone trabeculae. The alveolar ridge is not sharply

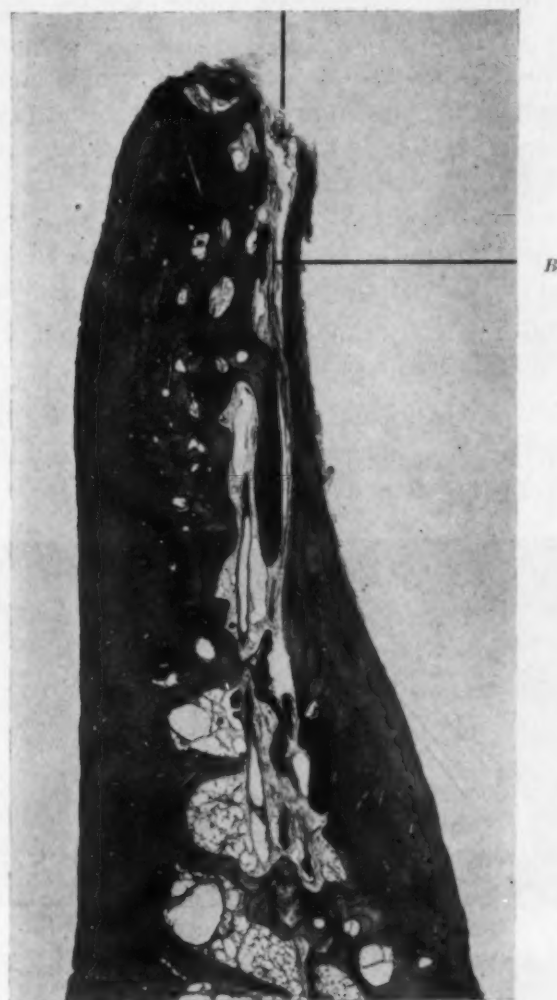


Fig. 18.—Enlargement of alveolar ridge of Fig. 17. *A*, Opening in alveolar ridge; *B*, circulatory channel with blood vessel. (Microsummar 42; distance from object $1\frac{1}{2}$ in.; extension of camera 21 in.; $\times 9.5$.) (Pl. 2036.)

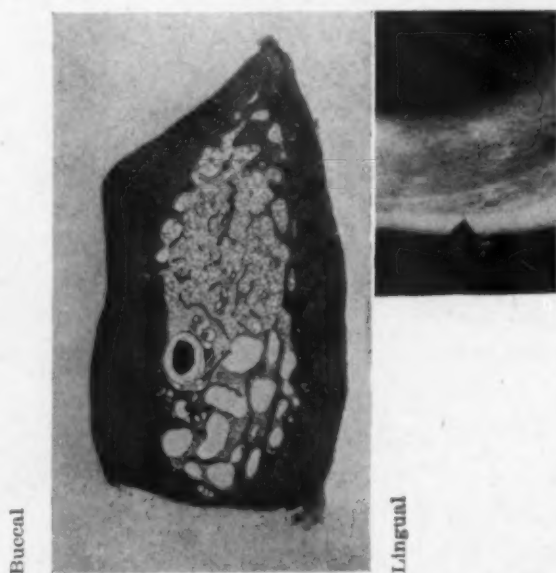


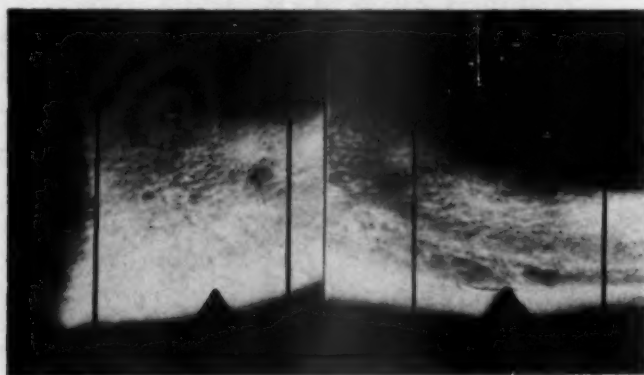
Fig. 19.—Jaw IV. Specimen 9. Buccolingual section through “^” in roentgenogram. (Microsummar 80; distance from object 3 in.; extension of camera 16 in.; $\times 3.5$.) (Pl. 5930, 5157.)

defined and the mandibular canal in the area over the nick is barely visible (Fig. 19).

Histologic Aspect.—The histologic specimen was obtained exactly through the nick which explains the flattening of the lower border of the mandible (Fig. 19). The cortical plate is slightly thicker on the buccal side than on the lingual; it is well ossified with a small number of marrow cavities. Between the two cortical plates a meshwork of small bone trabeculae can be seen with fat tissue in the marrow cavities. The two bony plates join at the crest in a pointed angle which explains why the height of the alveolar ridge does not appear sharply defined in the roentgenogram.

Jaw V

The fifth jaw was edentulous and without soft tissue. It was divided into Specimens 10 and 11 as indicated in Fig. 20 by the vertical lines. No data were available as to time of extraction of teeth or age of this individual.



(10)

(11)

Fig. 20.—Jaw V. (Edentulous.) Roentgenographic reproduction of the region from the lower left lateral incisor to the left third molar. Two specimens were obtained (Specimens 10 and 11) as indicated by the vertical lines. (Pl. 5155, 5158.)

SPECIMEN 10.—

Roentgenographic Aspect.—The specimen of this jaw again shows vertical radiolucent lines in the upper third of the mandible. The jaw seems to be well ossified with a heavy cortical layer at the lower margin. The mental foramen is clearly visible; however, the outline of the alveolar ridge is not distinct.

Histologic Aspect.—The labial cortical plate (Fig. 21) is slightly thinner than the lingual; they are connected by especially heavy crossbars (trabeculae). A fine network of supporting bone can be seen in the upper third of the specimen; the lingual cortical plate thins out toward the alveolar ridge, and is penetrated by a blood vessel which, as in the previous specimen, is responsible for the appearance of the vertical radiolucent lines in the roentgenogram. The alveolar ridge is rather pointed. The large marrow cavities are filled with fat tissue.

SPECIMEN 11.—

Roentgenographic Aspect.—Bone trabeculae are more sparse in this specimen. Alveolar ridge is not sharply defined (Fig. 22).

Histologic Aspect.—The lower border of the mandible is not shown completely because this section runs exactly through the nick and presents a different configuration of the mandible. The cortical plate is approximately of

even width; however, it can be noted that the area of former extraction has not been closed completely by osseous structure. Small fragments of bone trabeculae have filled in near the cortical bone.

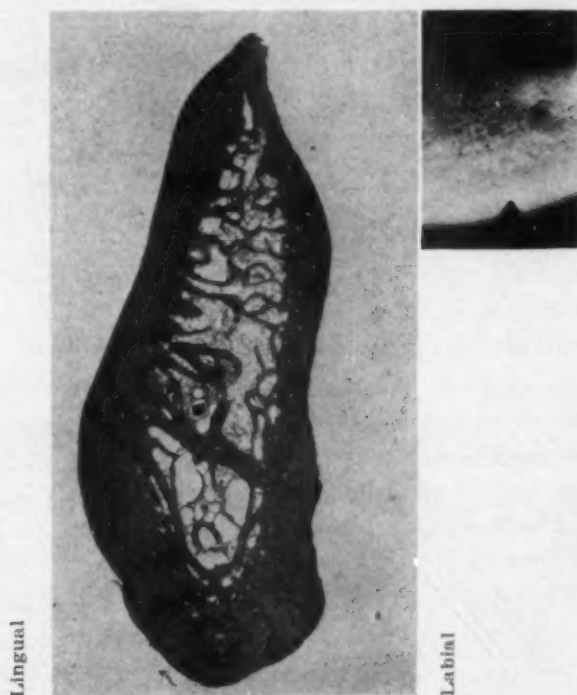


Fig. 21.—Jaw V. Specimen 10. Labiolingual section through “^” in roentgenogram. (Microsummar 80; distance from object 3 in.; extension of camera, 16 in.; $\times 3.5$.) (Pl. 5931, 5155.)

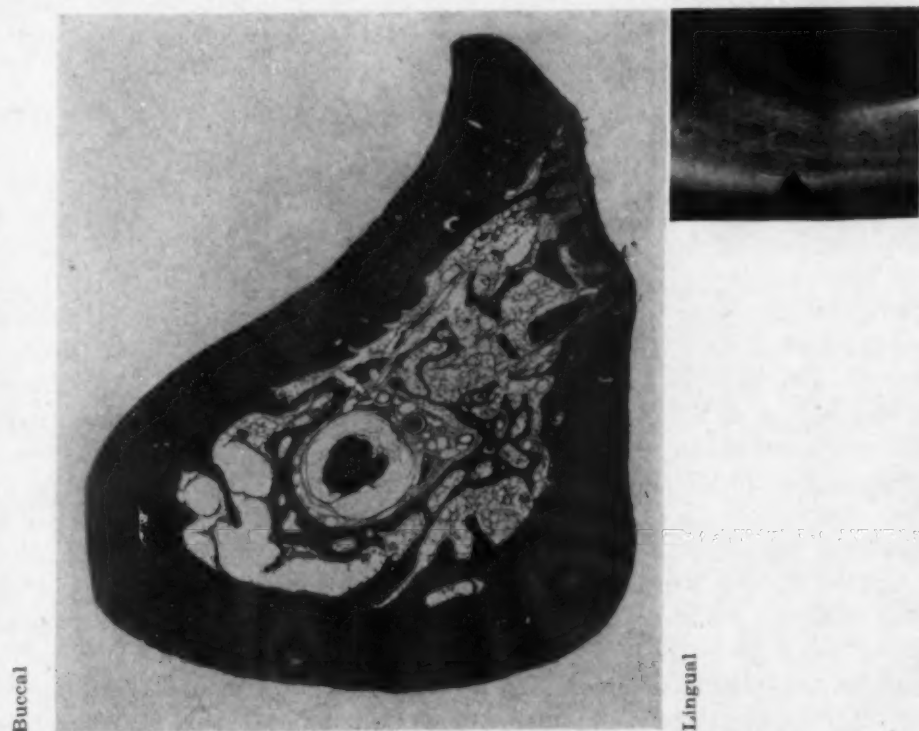


Fig. 22.—Jaw V. Specimen 11. Buccolingual section through “^” in roentgenogram. (Microsummar 80; distance from object 3 in.; extension of camera, 16 in.; $\times 3.5$.) (Pl. 1085, 5155.)

COMMENT AND CONCLUSIONS

Variations in the density, hardness, color, and texture of the osseous structures of the mandible are noted often in clinical practice. Roentgenograms of human jaws also show a variety of architectural designs which (from the standpoint of diagnosis, prognosis, and treatment of a given condition) present important criteria of exogenous or endogenous influences. Furthermore, it is generally recognized that the interpretation of dental roentgenograms is by no means uniform because considerable differences exist in the morphologic character of normal and pathologic structures. It must be realized that the diagnosis of a clinical condition should never be made on the basis of the roentgenographic data alone and that the roentgenogram represents only an aid in the diagnosis of normal or pathologic conditions. However, a sound understanding of the fundamentals of roentgen images greatly contributes to our diagnostic ability and thus to the establishment of sound therapeutic procedures.

The microscopic study of biopsy material is almost exclusively used for the diagnosis or confirmation of the clinical diagnosis of neoplastic tissues and probably represents the most accurate method for the analysis of tissue. There are innumerable factors which can influence the structures of the jaws which may be of exogenous as well as endogenous origin, of infectious or noninfectious nature. Metabolic disorders have been given little consideration because their specific effects and the pathogenesis of tissue lesions have not been correlated with conditions observed in dental practice. Experimental studies have emphasized the fact that it is possible to influence jawbone development in growing animals by alterations of diet, and several of the experiments have given information of the utmost value in the control of human bone diseases. In an effort to analyze critically the relationship between jawbone pattern (especially in osteoporosis and osteodystrophies), and general systemic disorders, it was soon found that bony architecture varies individually to such an extent that it was most difficult to interpret borderline cases.

How different the jawbone structure can appear in human dental roentgenograms is illustrated in Fig. 23.

The three roentgenographic reproductions in Fig. 23 were selected from the same areas in three different individuals of approximately the same age. In all three instances the first molar had been extracted. In examining the distributions of bone trabeculae in the area of the extracted molar it became immediately apparent that the bony replacement differs in each case. In every instance neither the buccal nor the lingual plate was removed, the course of healing was uneventful, and in two of the patients, Fig. 23, *B* and *C*, there was clinically slight hemorrhage of the gum tissue. Fig. 23, *A* shows a dense osseous pattern; the marrow cavities are practically all of the same size, a picture which is representative of all areas in the upper and lower jaws. In other words, following the extraction of the first molar the bone trabeculae filled in according to the general bony pattern of this individual which in this instance is of a normal type. In Fig. 23, *B* a regular distribution of bone trabeculae as seen in Fig. 23, *A* is missing. In fact, trabeculae can only be seen near the apical third of the second molar and close to the surface of the roots of this tooth. They radiate to some extent from these areas into the space formerly occupied by the first molar. Hardly any bone trabeculae can be seen in the entire alveolar ridge. This roentgenogram presents the typical picture of osteoporosis which was accompanied in this patient by a thyroid dysfunction. This osteoporotic pattern

in the first molar area is also characteristic for most areas of the upper and lower jaws with indications of progressive resorption of bone trabeculae. The local increase of bone trabeculae around the root apices of the second premolar and second molar must be interpreted as being the result of the functional stress increased by the presence of an artificial appliance. In contrast to Fig. 23, *A* and *B* a still different bone pattern is found in Fig. 23, *C*. A fine network of bone trabeculae is seen which seems to run primarily in a mesiodistal direction and in some areas the distribution appears denser than in others. This picture is representative of an osteodystrophy; the individual suffered from Paget's disease. As in Fig. 23, *B* an artificial appliance had been placed, and slightly mesial to the central area between the second premolar and second molar there is a circular radiolucent area, apparently the result of a residual infection. Hypercementosis can be seen on the surfaces of the roots of the first and second premolars which gives the roots a most irregular outline.

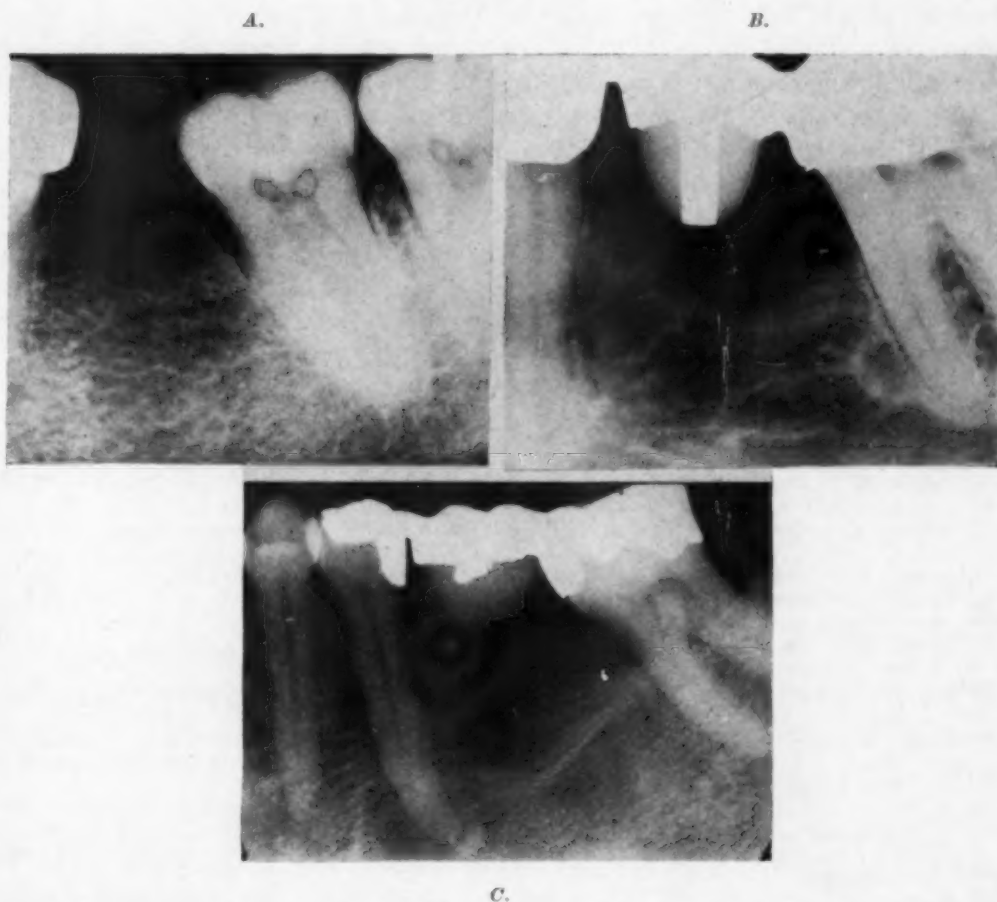


Fig. 23.—Roentgenographic reproductions of lower left first molar areas. *A*, Normal bone structure; *B*, progressive bone atrophy; *C*, osteodystrophy (Paget's disease). (Pl. 998, 1030, 1161.)

In order to bring out these differences in structural designs it is obvious that the highest type of roentgenographic technique including accurate angulation, exposure, and processing must be employed. The failure to recognize such outstanding pathologic conditions is due only too frequently to a lack of standardization of technique.

Through comparative roentgenographic studies it is not too difficult to gain sufficient experience in diagnosing these extreme forms of pathologic bone

conditions. However, difficulties arise in attempting to interpret mitigated forms and for this reason a comparative histologic and roentgenographic study was undertaken of human autopsy material in order to add to our knowledge of the true nature of structures seen in roentgenograms.

SUMMARY

Five lower jaws were studied and analyzed by comparing roentgenograms and histologic preparations. In all instances it was found that the roentgenograms confirmed the histologic aspect in every detail. If standardized methods of roentgenographic technique are employed, differences in the general shade of the images of jawbone tissue are primarily due to individual differences in density of ossification and combined thickness of the cortical plates. The number of bone trabeculae between the cortical plates and alveolar bone is not only dependent on the functional requirements which the jawbone has to meet, but also on the many general systemic conditions which affect bone metabolism, and control the laws of bone resorption and apposition. The jaw structures are by no means of a stabile nature, and as the trabeculae are a storehouse of important minerals upon which the body may draw in time of need, it must be emphasized that morphologic changes may occur rapidly at any time. They do so, however, in a characteristic manner, and knowledge of these changes from the standpoint of roentgenographic interpretation becomes an extremely important asset in dental practice.

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CHANGES IN THE ORAL MUCOSA ACCOMPANYING ACUTE PANTOTHENIC ACID DEFICIENCY IN YOUNG RATS

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INTRODUCTION

THE extreme oral changes observed by Becks, Wainwright, and Morgan² in dogs as a result of a prolonged filtrate fraction deficiency suggested further investigations of the specific factors producing those changes in a more rapidly growing species such as the rat. The earlier studies of Becks and Morgan¹ on younger dogs deficient in the filtrate fraction, and the production of noma in monkeys deficient in several members of the B₂ complex by Topping and Frazer¹⁰ had served to point out the fundamental importance to the oral epithelium of

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TABLE I. NUMBER AND PERCENTAGE OF ANIMALS DYING BY FIVE-DAY INTERVALS OF 226 RATS FED PANTOTHENIC ACID DEFICIENT DIETS FROM DAY 1

| AGE (DAYS) | f | PER CENT BY 5-DAY INTERVALS | PER CENT CUMULATIVE |
|---------------|-----|--------------------------------|------------------------|
| 1- 5 | 12 | 5.3 | 5.3 |
| 6- 10 | 7 | 3.1 | 8.4 |
| 11- 15 | 14 | 6.2 | 14.6 |
| 16- 20 | 11 | 4.9 | 19.5 |
| 21- 25 | 17 | 7.5 | 27.0 |
| 26- 30 | 33 | 14.7 | 41.7 |
| 31- 35 | 34 | 15.1 | 56.8 |
| 36- 40 | 34 | 15.1 | 71.9 |
| 41- 45 | 17 | 7.5 | 79.4 |
| 46- 50 | 10 | 4.4 | 83.8 |
| 51- 55 | 4 | 1.8 | 85.6 |
| 56- 60 | 7 | 3.1 | 88.7 |
| 61- 65 | 10 | 4.4 | 93.1 |
| 66- 70 | 4 | 1.8 | 94.9 |
| 71- 75 | 3 | 1.3 | 96.2 |
| 76- 80 | 2 | 0.9 | 97.1 |
| 81- 85 | 1 | 0.4 | 97.5 |
| 86- 90 | 0 | 0 | 97.5 |
| 91- 95 | 3 | 1.3 | 98.8 |
| 96-100 | 1 | 0.4 | 99.2 |
| 101-105 | 1 | 0.4 | 99.6 |
| 106-110 | 1 | 0.4 | 100.0 |
| Total | 226 | 100.0 | |

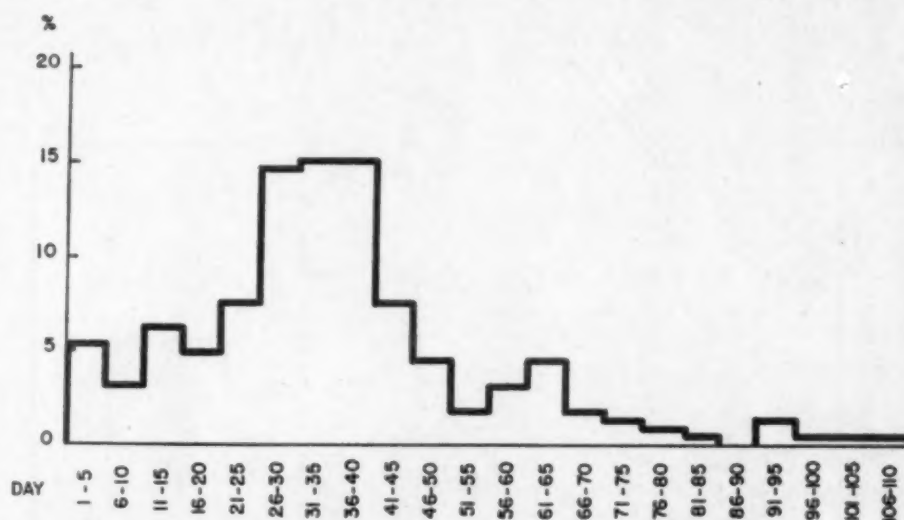


Fig. 1.—Percentage of animals dying by five-day intervals of 226 rats fed pantothenic acid deficient diets from day 1.

the filtrate fraction,* in which pantothenic acid is the principal factor. In the dermatological lesions of pantothenic acid deficiency in the rat, Sullivan and Nichols⁹ have noted a lack of inflammatory response similar to that described in the oral lesions.²

Nelson and others^{6, 11, 12} have shown that in the rat purified diets supplemented with the known crystalline B vitamins except biotin and folic acid are adequate for growth, reproduction, and lactation in contrast to their inadequacy for other species. In the dog, for example, maintenance on highly purified diets requires additional unknown vitamins.^{3, 4, 8} This makes it possible to devise a purified diet for the rat which is a closer approach to an uncomplicated pantothenic acid deficiency.⁷

*The term "filtrate fraction" included pantothenic acid and unknown factors of the vitamin B complex; it excluded thiamin, riboflavin, pyridoxine, and nicotinic acid. According to the present knowledge of vitamins the term would include pantothenic acid, inositol, p-aminobenzoic acid, choline, biotin, folic acid, and unknowns.

TABLE II. OCCURRENCE OF DERMATITIS AND GRAYING OF HAIR IN 226 RATS FED PANTOTHENIC ACID DEFICIENT DIETS FROM DAY 1

| AGE (DAYS) | NUMBER OF SURVIVORS | DERMATITIS | | GRAYING OF HAIR | |
|---------------|------------------------|------------|-------------------|-----------------|-------------------|
| | | NUMBER | % OF SURVIVORS | NUMBER | % OF SURVIVORS |
| 5 | 214 | 0 | 0 | 0 | 0 |
| 10 | 207 | 0 | 0 | 0 | 0 |
| 15 | 193 | 0 | 0 | 0 | 0 |
| 20 | 182 | 0 | 0 | 0 | 0 |
| 25 | 165 | 0 | 0 | 0 | 0 |
| 30 | 132 | 0 | 0 | 0 | 0 |
| 35 | 98 | 2 | 2.0 | 0 | 0 |
| 40 | 64 | 5 | 7.8 | 0 | 0 |
| 45 | 47 | 8 | 17.0 | 0 | 0 |
| 50 | 37 | 10 | 27.0 | 0 | 0 |
| 55 | 33 | 13 | 39.4 | 0 | 0 |
| 60 | 26 | 17 | 65.4 | 0 | 0 |
| 65 | 16 | 11 | 68.8 | 0 | 0 |
| 70 | 12 | 8 | 66.7 | 2 | 20.0* |
| 75 | 9 | 7 | 77.8 | 1 | 14.3* |
| 80 | 7 | 7 | 100.0 | 1 | 20.0* |
| 85 | 6 | 6 | 100.0 | 2 | 50.0* |
| 90 | 6 | 6 | 100.0 | 2 | 50.0* |
| 95 | 3 | 3 | 100.0 | 1 | 50.0† |
| 100 | 2 | 2 | 100.0 | 0 | 0 |
| 105 | 1 | 1 | 100.0 | 0 | 0 |
| 110 | 0 | | | | |
| Of Total | | 35 | 15.5 | 3 | 1.3 |

*The groups from 70 to 90 days, each included two white rats; therefore the percentage incidence of gray hair was calculated on the basis of the number of survivors less 2.

†The group at 95 days contained one white rat.

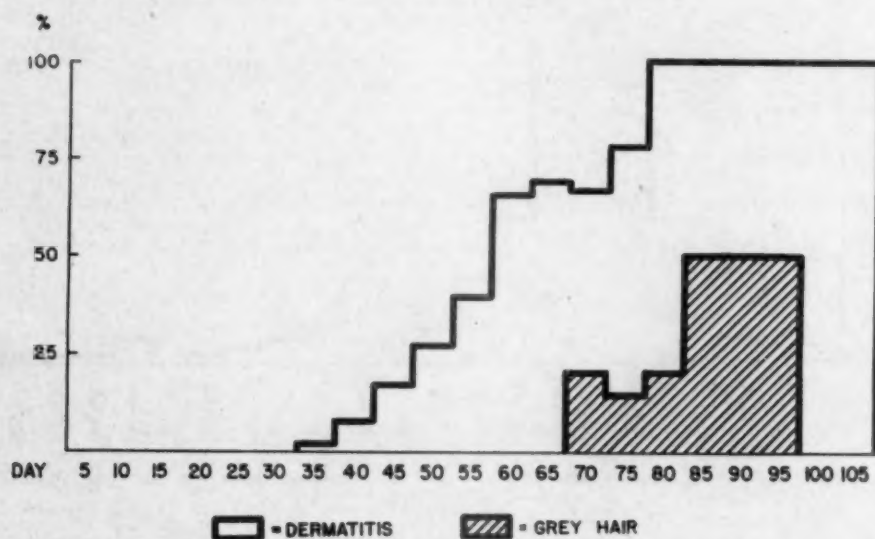
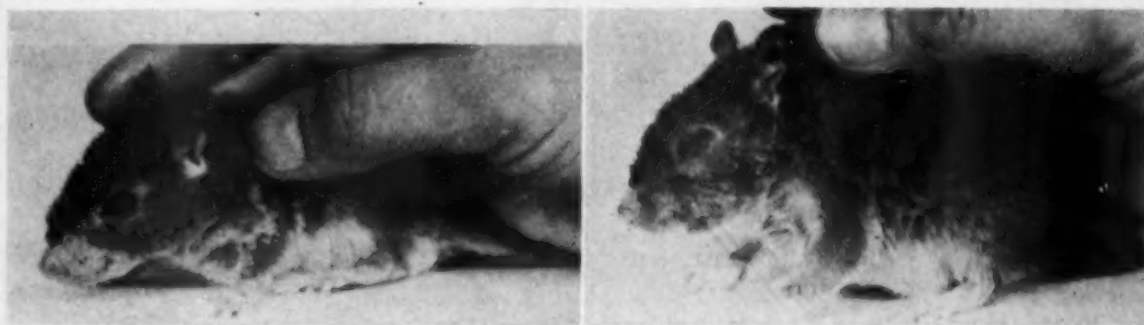


Fig. 2.—Occurrence of dermatitis and graying of hair in 226 rats fed pantothenic acid deficient diets from day 1. Calculated as percentage of survivors, by five-day intervals.

In an investigation of the syndrome of pantothenic acid deficiency in the rat by Nelson⁷ it was found that the most acute deficiency could be produced by placing normal litters on the deficient diet on the day of birth. This is in contrast to the usual procedure in which rats are placed on experimental diets the day of weaning, day 21. Such young suckling rats were severely retarded in growth and survived only a short time. For the majority of the young, as seen in Fig. 1, the most critical period of the deficiency occurred between 25 and 40 days of age. The numbers of animals and the percentages by five-day intervals are found in Table I which shows that at 20 days 19.5 per cent had died, at 60 days 88.7 per cent, and at 75 days 96.2 per cent. The earliest death occurred at 5 days of age and the oldest animal survived only for 109 days. The de-

iciency was so acute that many of the animals died before 30 days of age severely retarded in growth⁷ but without developing any other deficiency symptoms. In the animals that survived a longer period of time, the characteristic symptoms of the deficiency (porphyrin deposition⁵ and dermatitis) gradually appeared, usually after 45 days of age. Graying and dermatitis, typical deficiency symptoms in older rats, were rarely seen before 45 days of age (Fig. 2). The earliest appearance of dermatitis occurred at 35 days while graying of hair appeared later at 70 days and less frequently (Fig. 2). Only 35 of the 226 rats developed dermatitis and only 3 showed graying of hair (Table II). Although these incidences are low numerically, by the end of the experiment there were so few animals alive that 100 per cent of them showed dermatitis, and 50 per cent, graying of hair. Typical examples of the dermatitis and "spectacled eyes" are seen in the rats of Fig. 3. Note the poor condition and marked retardation of growth as compared with the litter mate control, Fig. 4, which received calcium pantothenate.



A.

B.

Fig. 3.—Dermatitis and marked retardation of growth in gray rats deficient in pantothenic acid from birth. Note the loss of hair in the inflamed areas on the nose and around the eyes ("spectacled eye" condition). The rats of Figs. 3 and 4 are litter mates and the photographs are of identical magnification.

A, Age 66 days, weight 36 grams (Rat G3868♂, Pl. 20029K). B, The eyes are practically closed with crusted exudate. Age 66 days, weight 53 grams (Rat G3869♂, Pl. 20032K).



Fig. 4.—Excellent condition and growth in pantothenic acid control black rat. Age 66 days, weight 271 grams (Rat B3872♂, Pl. 20022K).

EXPERIMENTAL ARRANGEMENT AND CONTENT OF DIETS

Oral and dental changes in the animals of Nelson⁷ were studied in three groups of rats. The first consisted of 17 animals (Table III, Col. 3) which had been placed on a pantothenic acid deficient diet *at birth*. For comparison, a

second group of four rats was examined which had been placed on a pantothenic acid deficient diet at the age of weaning, *day 21* (Table IV, Col. 3). The third group of 5 rats was also started at *day 21* but a high level of fats was fed (Table IV, Col. 3) in contrast to the usual high level of carbohydrates.

TABLE III. TYPES OF DIETS, DISTRIBUTION AND AGES OF ANIMALS FED PANTOTHENIC ACID DEFICIENT DIETS FROM DAY 1

| (1) AGE AT AUTOPSY* (DAYS) | (2) SEX | (3) PANTOTHENIC ACID DEFICIENT | | (4) PURIFIED DIET CON- TROLS, RECEIVED CAL- CIUM PANTOTHENATE FROM BIRTH | | (5) NORMAL CONTROLS STOCK DIET 1A PLUS LETTUCE | |
|-------------------------------------|------------|--------------------------------------|-----------|--|-----------|---|-----------|
| | | RAT NO. | SPEC. NO. | RAT NO. | SPEC. NO. | RAT NO. | SPEC. NO. |
| 21 | ♀ | BH3522 | 7904 | B8746 | 7927 | BH5440 | 7929 |
| 26 | ♂ | B8734 | 7905 | B8744 | 7928 | BH8435 | 7930 |
| 30 | ♀ | BH9133 | 7906 | W7031 | 7916 | B4530 | 7931 |
| 33 | ♂ | B5199 | 8622 | B5423 | 8623 | | |
| 34 | ♂ | BH9132 | 7907 | GH6931 | 7917 | W8432 | 7932 |
| 36 | ♂ | B8710 | 7908 | SH6716† | 7919 | B6754 | 7933 |
| 38 | ♂ | BH7050 | 7909 | BH6309 | 7920 | B4540 | 7934 |
| 45 | ♂ | SH6913 | 7910 | SH6914† | 7921 | B4915 | 7935 |
| 48 | ♀ | G4262 | 7911 | G4263† | 7922 | BH6336 | 7936 |
| 61 | ♂ | GH6901 | 7912 | G7718† | 7923 | G8470 | 7937 |
| 63 | ♀ | BH6902 | 7913 | B5015† | 7924 | G8442 | 7938 |
| 65 | ♂ | B4215 | 7914 | B4219† | 7925 | B17 | 7939 |
| 70 | ♂ | G3869 | 8625 | BH3873 | 8628 | W6126 | 8629 |
| 70 | ♂ | G3868 | 8626 | B3872 | 8627 | | |
| 72 | ♂ | B7020 | 7915 | B6315 | 7926 | B6326 | 7940 |
| 97 | ♂ | B8749 | 8577 | GH8767 | 8578 | B1132 | 6579 |
| 109 | ♀ | B3303 | 8580 | B5426 | 8581 | BH1136 | 8582 |
| No. of Animals | | 17 | | 17 | | 15 | |

*The experiments were conducted from August, 1942 to April, 1944.

†Pantothenic acid deficient: day 1 to 21; given calcium pantothenate day 21 on.

TABLE IV. TYPES OF DIETS, DISTRIBUTION AND AGES OF ANIMALS FED PANTOTHENIC ACID DEFICIENT DIETS FROM DAY 21

| TYPE OF DIET | (1) AGE AT AUTOPSY* (DAYS) | (2) SEX | (3) PANTOTHENIC ACID DEFICIENT | | (4) PURIFIED DIET CON- TROLS, RECEIVED CALCIUM PANTO- THENATE | | (5) NORMAL CONTROLS STOCK DIET 1A PLUS LETTUCE | |
|------------------------|-------------------------------------|------------|--------------------------------------|-----------|---|-----------|---|-----------|
| | | | RAT NO. | SPEC. NO. | RAT NO. | SPEC. NO. | RAT NO. | SPEC. NO. |
| High Carbo- hydrate | 74 | ♀ | B6114 | 8610 | BH6117 | 8611 | B6152 | 8612 |
| | 77 | ♂ | B4902 | 8616 | B4906 | 8618 | B4904 | 8617 |
| | 104 | ♀ | G4627 | 8615 | G5628 | 8613 | G5625 | 8614 |
| | 113 | ♂ | B5074 | 8619 | B5073 | 8620 | W5071 | 8621 |
| High Fat | 75 | ♀ | B5057 | 8595 | BH5060 | 8596 | B5058 | 8597 |
| | 88 | ♀ | BH4009 | 8598 | BH4012 | 8599 | B4008 | 8600 |
| | 117 | ♀ | B5668 | 8601 | B5671 | 8602 | B5672 | 8603 |
| | 129 | ♀ | B5621 | 8604 | B5620 | 8605 | B5624 | 8604 |
| | 157 | ♀ | B4017 | 8607 | B4019 | 8608 | B4021 | 8609 |

*The experiments were conducted from October, 1943, to December, 1944.

All rats were sacrificed as late as possible before death. Each was moribund and it was assumed that the most severe changes possible had taken place. Control animals receiving the purified diet supplemented with calcium pantothenate (Tables III and IV, Col. 4) and normal animals (Tables III and IV, Col. 5) maintained on natural food diets* were sacrificed at the same age as each deficient animal. The skulls of these animals were dissected, cut in half, fixed in 10 per cent formol, decalcified in 5 per cent aqueous solution of nitric acid, dehydrated, embedded in nitrocellulose and sectioned at 8 microns.

*The natural food diet (Diet 1A plus lettuce) was composed of ground whole wheat 67.5 per cent, casein 15 per cent, whole milk powder 10 per cent, hydrogenated cottonseed oil (Crisco) 5.25 per cent, calcium carbonate 1.5 per cent, and sodium chloride 0.75 per cent. Sufficient vitamin A-D concentrate (Sardilene) was added to furnish 100 U.S.P. units vitamin A and 14 chick units vitamin D per 10 grams diet.

Rats Deficient From Birth.—Seventeen rats, from a total of 226, were examined histologically, ranging in age at autopsy from 21 to 109 days (Table III, Col. 1). The deficient diet* was composed of alcohol-extracted casein 24 per cent, sucrose 63 per cent, McCollum salts No. 185, 4 per cent, and hydrogenated cottonseed oil (Crisco) 8 per cent. To each kilogram of diet was added a 20 per cent alcoholic solution of synthetic B vitamins that furnished 20 μ g thiamine, 20 μ g pyridoxine, 50 μ g p-aminobenzoic acid, 100 μ g nicotinic acid, and 5 mg. choline chloride per 10 grams of diet. A dry mixture of sucrose, riboflavin, and inositol was added that furnished 50 μ g riboflavin and 2 mg. inositol per 10 grams of diet. During lactation the mother of each litter, both deficient and control, received 1 c.c. fat-soluble vitamins weekly, and after weaning each individual rat was given 0.5 c.c. fat-soluble vitamins weekly by stomach tube. Each cubic centimeter of this fat-soluble vitamin mixture contained 650 mg. corn oil, 750 U.S.P. units vitamin A, 100 chick units vitamin D, and 6 mg. synthetic alpha-ocopherol. The control animals on purified diets (Table III, Col. 4) received calcium pantothenate with the purified diet starting from birth in the proportion of 280 μ g per 10 grams of diet; in six cases 500 μ g calcium pantothenate per 10 grams of diet was given daily to each rat from the day of weaning and none before as noted in Table III, Col. 4 and footnote 2.

Rats Deficient From Weaning.—*High carbohydrate diet:* Four rats (Table IV, Col. 3) were examined histologically from a group of 23 which were started on the deficiency at 21 days of age. At autopsy they ranged in age from 74 to 113 days (Table IV, Col. 1). These animals were fed a purified diet containing a higher level of B vitamins than that given the previous group of animals, a different salt mixture, and the usual high proportion of carbohydrate in the form of sucrose. The deficient diet contained alcohol-extracted casein 24 per cent, sucrose 64 per cent, Elvehjem salts No. 4, 4 per cent, and hydrogenated cottonseed oil (Crisco) 8 per cent. To each kilogram of diet was added a dry mixture of sucrose (included in the percentage given above) with synthetic B vitamins that will furnish 50 μ g thiamin, 50 μ g pyridoxine, 100 μ g riboflavin, 100 μ g p-aminobenzoic acid, 200 μ g nicotinic acid, 4 mg. inositol, and 10 mg. choline chloride per 10 grams of diet. The amount of fat-soluble vitamins was the same as that given the day 1 deficient group. The control animals on the purified diet received 500 μ g of calcium pantothenate per 10 grams of diet (Table IV, Col. 4).

High fat diet: Five rats (Table IV, Col. 3) were examined histologically from a group of 13 which also had been started on the deficiency on day 21 but which received a very high proportion of their caloric intake from fats. The ages at autopsy ranged from 75 to 157 days. The diet was composed of alcohol-extracted casein 24 per cent, sucrose 24 per cent, Elvehjem salts No. 4, 4 per cent, hydrogenated cottonseed oil (Crisco) 48 per cent. The vitamin levels were the same as those given the day 21 deficient and control, high carbohydrate groups.

RESULTS AND DISCUSSION

Rats Deficient From Birth.—In the youngest deficient rats, from 21 to 45 days of age, the paradental tissues about the lower molar teeth presented an ordinary appearance in general although hyperkeratosis of the epithelium was marked and extended beyond the areas covering the erupting teeth. In control

*There were slight variations in the preparation of the casein and the content of the salt mixture which are noted separately.¹ No effect was found from these factors, probably because of the short period of the experiment.

animals on the purified diet, the molar teeth had erupted earlier¹³ and the surface epithelium was normal in appearance.

Increased desquamation of the gingival epithelium between the lower second and third molars in a 61-day-old deficient rat is seen in Fig. 5. Note the early proliferation of the epithelium at the basal layer. In another young deficient rat, 48 days old, hyperkeratosis and necrosis (at *A* and *B*, Fig. 6) has begun to involve the epithelium beyond the normal level of the gingival crest. Until this age the eruption of the molar teeth has been incomplete and much of the tissue change has been overshadowed by the destruction incident to eruption. In addition to surface changes, the epithelium is proliferating rapidly as seen at *C*, Fig. 6. There is no evidence of inflammation in the underlying connective tissue.

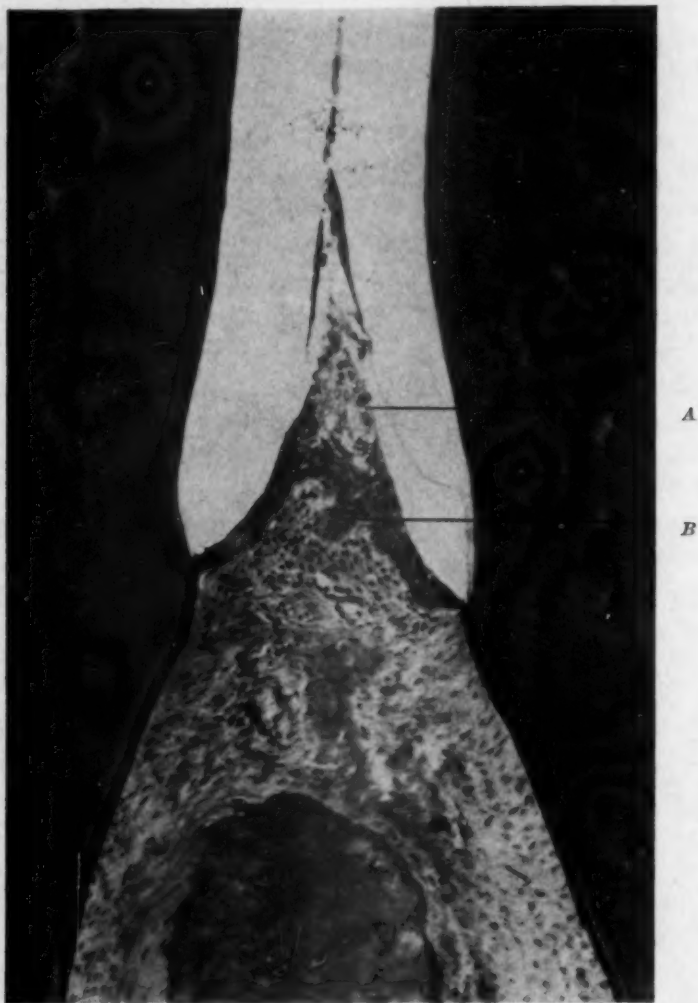


Fig. 5.—Increased desquamation of epithelium (*A*) between lower second and third molars of a pantothenic acid deficient male rat aged 61 days. Note proliferation of epithelium (*B*) and lack of inflammatory response. (Rat GH6901, Spec. 7912, Pl. 8132.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 10; extension of camera 103 cm.; $\times 252$.)

Examination of a similar area between the lower second and third molars in older rats discloses beginning necrosis of the epithelium. An example is shown in Fig. 7 where necrosis has advanced halfway through the epithelium in a 63-day-old rat. Marked hyperkeratosis and necrosis were seen in other areas of the gingival tissue such as those on the mesial side of the lower first molar of this animal (Fig. 8). Necrosis of epithelium was observed regularly in the

interdental papilla of deficient animals; a deeper penetration is seen in a 72-day-old animal in Fig. 9. In these animals the basal cells of the epithelium have been able to proliferate rapidly enough to keep ahead of the advancing necrosis and thereby maintain a protective covering for the underlying connective tissues.

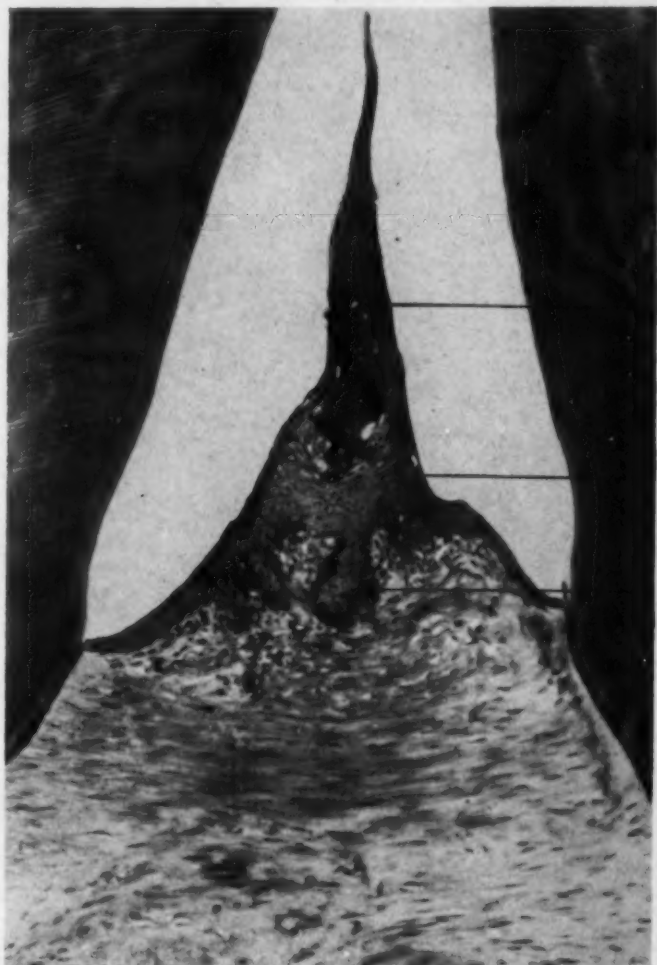


Fig. 6. —Degeneration of oral epithelium with hyperkeratosis and necrosis of epithelium at A and B, between the lower second and third molars of a pantothenic acid deficient 48-day-old female rat. Note proliferation of epithelium (e.g., at C) and the absence of inflammatory changes in the underlying connective tissue. (Rat G4262, Spec. 7911, Pl. 1975.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 10; extension of camera 103 cm.; $\times 252$.)

The most advanced stages of gingival necrosis observed in rats on a pantothenic acid deficiency are seen in Figs. 11 and 12. In these animals, aged 72 and 109 days, the necrotic process has advanced beyond the level of the cemento-enamel junction and yet inflammatory symptoms are absent. Not a single vitally staining epithelial cell is to be seen in Fig. 11 where the necrotic process has advanced to the margin of the alveolar bone, and in Fig. 12 the bony alveolar crest is below its normal level. The interdental papilla of the 72-day-old control animal receiving the same purified diet *plus* pantothenic acid is seen in Fig. 10. The epithelium is almost normal, appearing as a narrow band covering the papilla and showing but slight proliferation. Evidence of the wide spread of the changes due to the deficiency is seen in Fig. 13 in which one of the *palatine* ridges is shown of the 72-day-old deficient animal. Necrosis has advanced deeply through the epithelium and underlying connective tissue to a depth of nearly a millimeter, 100 times the normal thickness of the oral epithelium at this point.

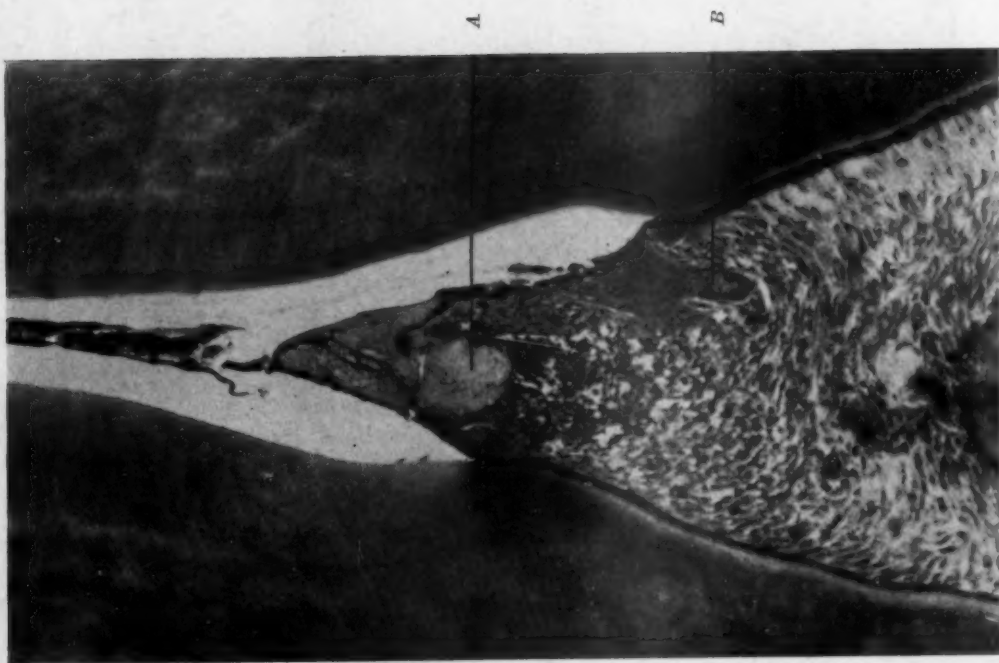


Fig. 7.

Fig. 7.—Rapidly advancing necrosis of epithelium (at A) in a pantothenic acid deficient female rat 63 days of age. Note proliferation of epithelium at B and lack of inflammatory response in underlying connective tissue. (Rat BH6902, Spec. 7913, Pl. 1978.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 10; extension of camera 103 cm.; $\times 252$.)

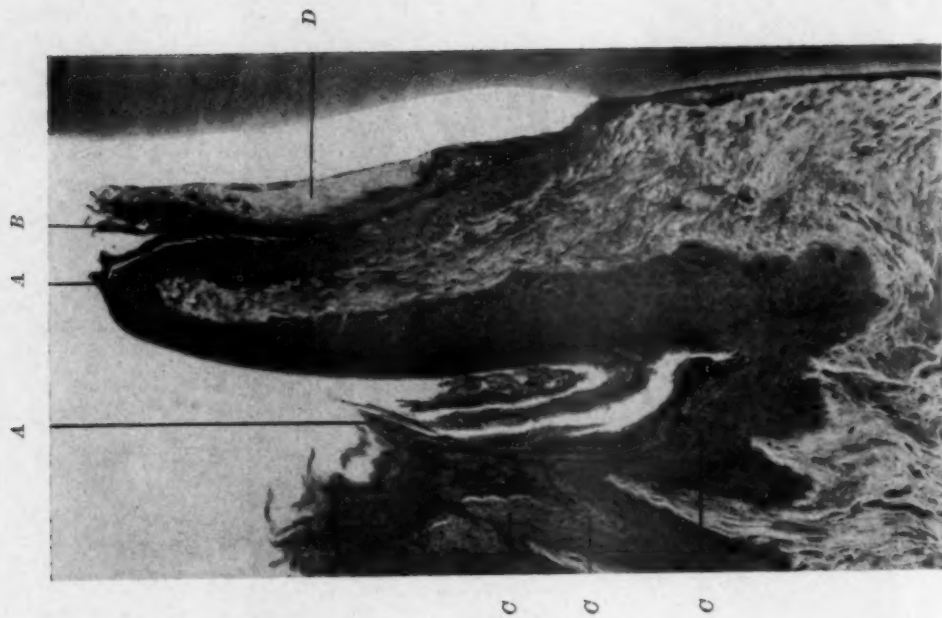


Fig. 8.

Fig. 8.—Hyperkeratosis of mouth epithelium (A) and enamel epithelium (B) and necrosis of mouth epithelium (C) and enamel epithelium (D) at the mesial surface of the lower first molar. Note the lack of inflammatory signs. This female rat aged 63 days is the same pantothenic acid deficient animal shown in Fig. 7. (Rat BH6902, Spec. 7913, Pl. 963.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 10; extension of camera 103 cm.; $\times 252$.)

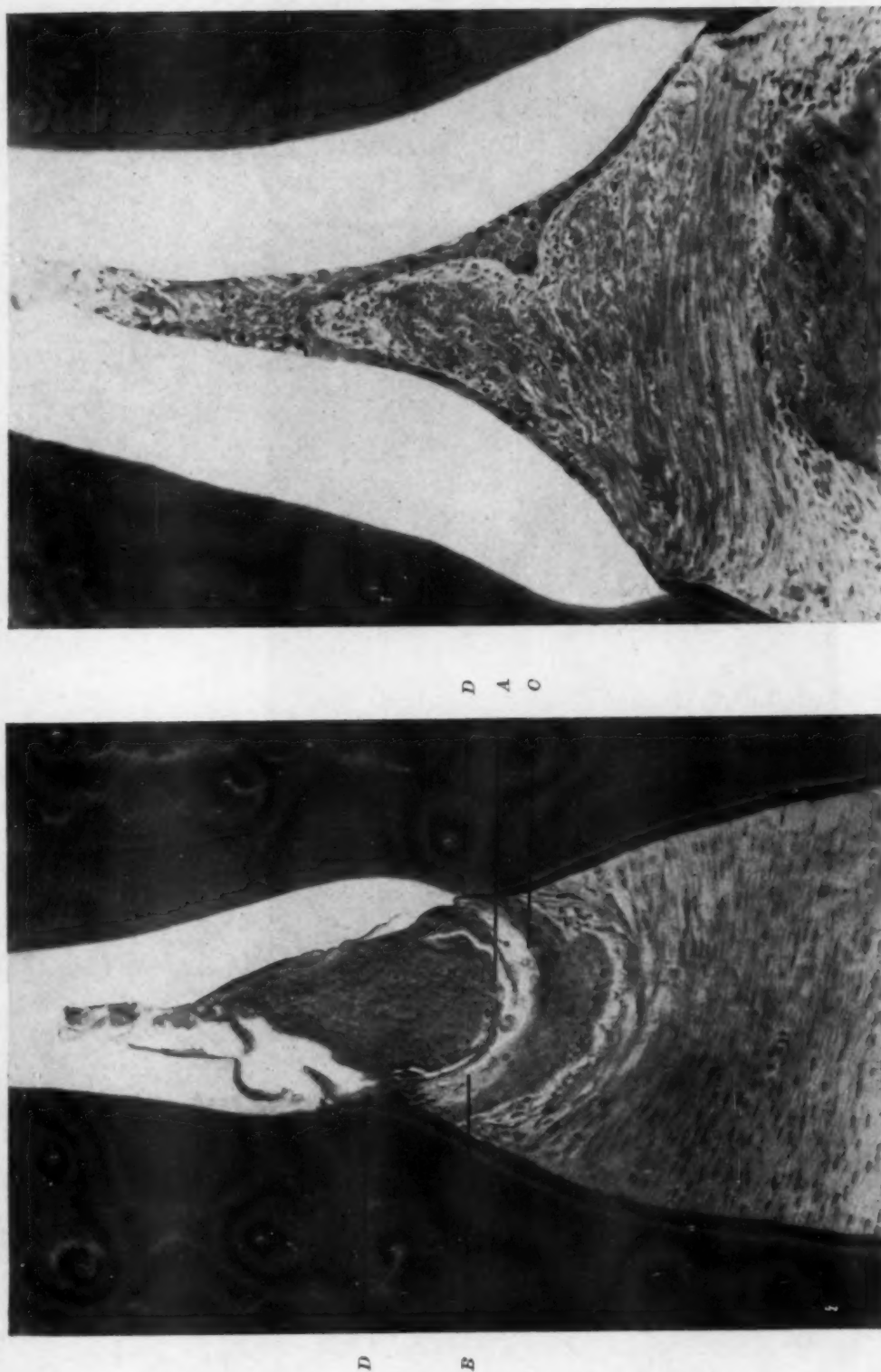


Fig. 9.

Fig. 9.—Advanced necrosis of epithelium down to A with beginning karyorrhexis at B and a few vital (well-stained) cells at C, between the lower first and second molars of a pantothenic acid deficient male rat 72 days of age. All of these epithelial cells are newly proliferated below the cemento-enamel junction (D) (compare with Fig. 5). Note the absence of inflammatory response. (Rat B7020, Spec. 7915, Pl. 8131.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 10; extension of camera 103 cm.; $\times 252$.)

Fig. 10.—Interstitial papilla of control rat, 72 days of age, receiving pantothenic acid in the purified diet. (Rat B6315, Spec. 7926, Pl. 8442.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 10; extension of camera 103 cm.; $\times 252$.)

In the oral epithelium no histologic differences were found in these day 1 deficient animals with regard to sex or the variations in the deficient diets. The typical necrosis and lack of inflammatory response were not seen in the control rats receiving either the purified diets or the natural food diets. In pointing out the consistent lack of inflammatory response it must be emphasized that this is not observed in a deficiency of niacin, as seen in dogs.² In the case of niacin deficiency, although it is also a member of the B group of vitamins, marked inflammatory symptoms appear, including hyperemia and edema of the tongue and oral mucosa.



A

Fig. 11.—Complete necrosis of epithelium and advanced necrosis of underlying connective tissue down to the crest of the interdental bone at A, between the lower second and third molars of the 72-day-old male rat of Fig. 7. Not a single vital epithelial cell is to be seen in this section where the necrosis has invaded far beyond the cemento-enamel junction (B), while characteristically in this deficiency of pantothenic acid, there is a lack of inflammatory response. (Rat B7020, Spec. 7915, Pk 1974.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 10; extension of camera 103 cm.; $\times 252$.)

Rats Deficient From Weaning.—High carbohydrate diet: The severity and consistent nature of the damage noted in pantothenic acid deficiency are confirmed upon comparing the changes just observed in the acute deficiency with those in the animals placed on the deficient diet at day 21. Marked dermatitis, retardation of growth, and general poor condition with hair changes are seen in Fig. 14, A. In these rats, which survived a longer period, the usual necrosis



Fig. 12.—Advanced necrosis of epithelium to A with a thin layer of epithelial cells immediately below, between the second and third molars of a pantothenic acid deficient female rat 109 days of age. The epithelium is attached to the root surface at a point, B, far below the cemento-enamel junction, C. Note the absence of inflammatory response. (Rat B3303, Spec. 8580, Pl. 8370.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 10; extension of camera 103 cm.; $\times 252$.)

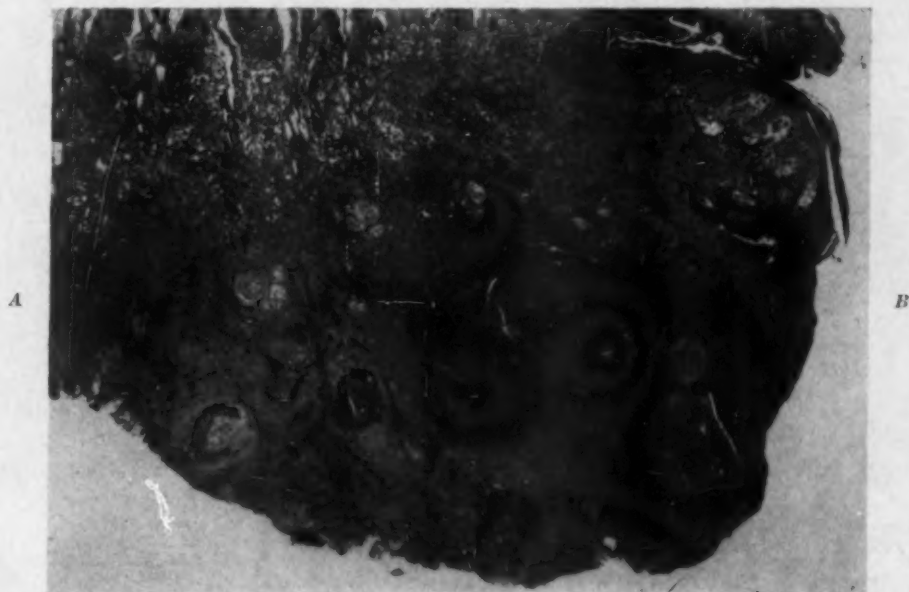


Fig. 13.—Necrosis of the first prominence (normally found) in the palate of the rat immediately distal to the upper incisor of the 72-day-old animal of Figs. 7 and 9. The necrotic area extends from the oral cavity upward to the line A.B. (Rat B7020, Spec. 7915, Pl. 1976.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal II, apochromatic 6; extension of camera 103 cm.; $\times 97$.)

of oral epithelium and proliferation of basal cells were observed. In an animal 119 days of age the entire tip of the tongue was destroyed as well (Fig. 15, A and B). An area from the inferior surface near the margin of the lingual ulcer is seen in Fig. 15, C. Note the lack of edema which would be expected in such severe destruction. The chronic nature of the lesion is also evidenced by the tenacity of the crust formed by the exuded fluids. Epithelial hyperplasia was seen frequently in the gingival tissues of these animals in the form of spurs from the surface epithelium and enlargements of Malassez's rests; their appearance closely resembled the changes observed earlier in dogs deficient in pantothenic acid.



Fig. 14.—Marked dermatitis of nose, retardation of growth and poor general condition in black rats deficient in pantothenic acid from weaning.

A, High-carbohydrate diet. Note the dun-colored fur with the lack of long guard hairs. Age 88 days, weight 120 grams. (Rat B5074♂, Pl. 20060M.)

B, High-fat diet. Note the graying and "spectacled eyes." Age 128 days, weight 69 grams. (Rat B5621♀, Pl. 20018K.)

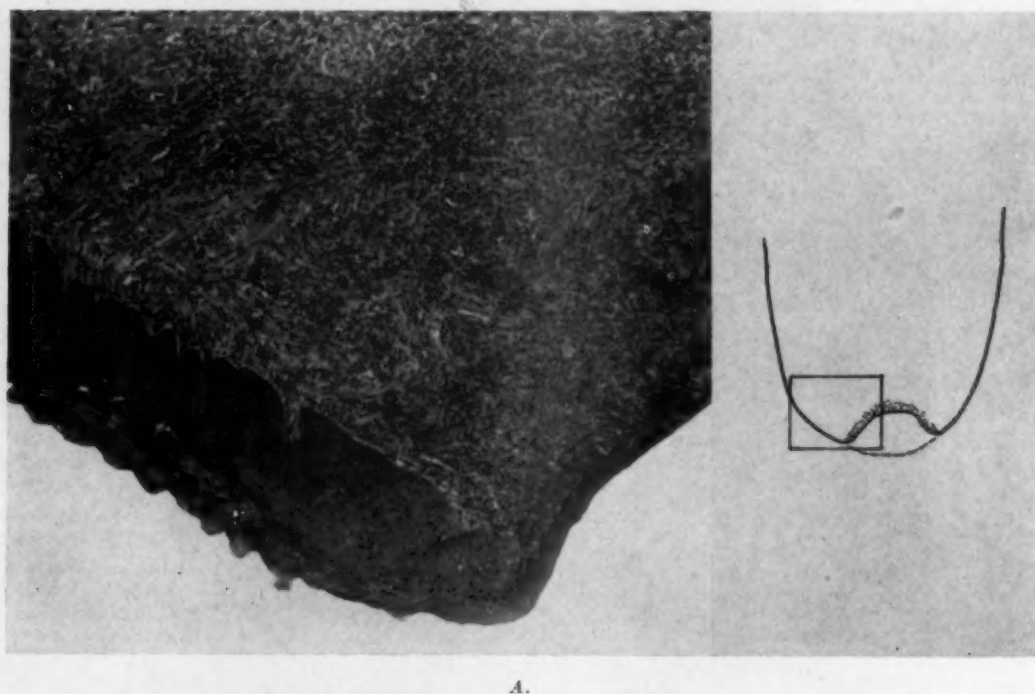
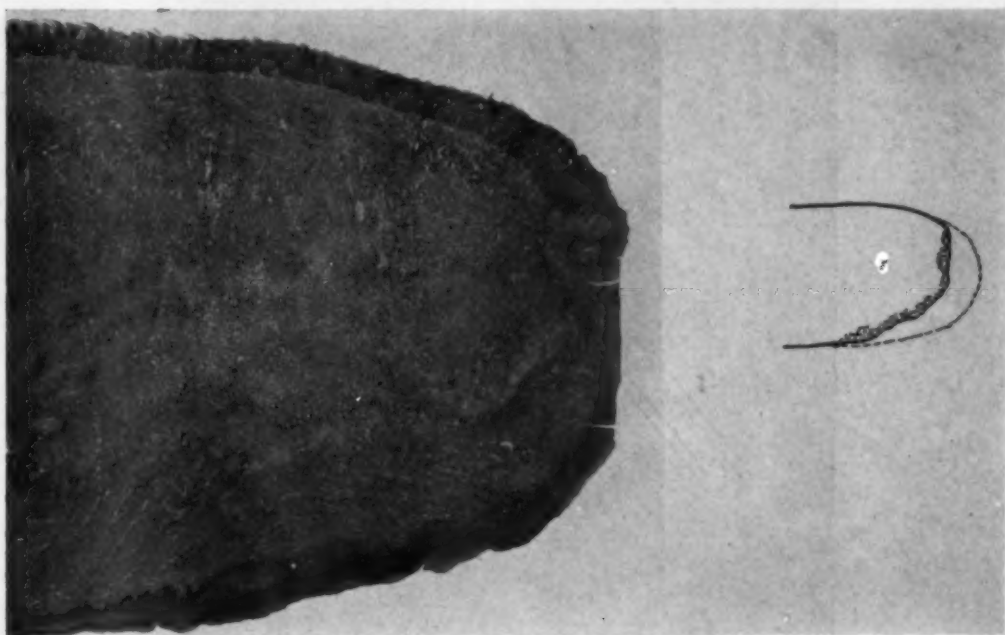


Fig. 15.—Large ulcer (2 by 4 mm.) which had destroyed the tip of the tongue in a pantothenic acid deficient male rat 113 days of age. Note the lack of swelling in the presence of great destruction. (Rat B5074, Spec. 8619.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin.)

A, Border of lingual ulcer in horizontal section; sketch shows original contours and area of section. (Pl. 8512.) (Zeiss Homal VI, apochromatic 6; extension of camera 80 cm.; X114.)

In noting this confirmation of the changes in pantothenic acid deficiency it is to be pointed out that this group of animals developed the changes in spite of deferring the start of the deficiency until the age of weaning. Furthermore, the basal diet was superior as it contained increased amounts of all other members of the B vitamin group and an improved salt mixture.

High fat diet: Since vitamin requirements are often influenced by the proportions of nutrients another group of weanling rats were fed a diet similar to the previous but with 48 per cent of the food in the form of fat. Typical dermatitis developed as seen in Fig. 14, *B*. These animals likewise survived



B.



C.

Fig. 15 (Continued).

B, Sagittal section showing wide destruction at the tip and inferior surfaces of the tongue; sketch illustrates original outline by broken line. (Pl. 8513.) (Zeiss Microtar f=2 cm.; extension of camera 50 cm.; $\times 25$.)

C, Area near border of ulcer, from inferior surface, showing crusted exudate. (Pl. 8371.) (Zeiss Homal II, apochromatic 6; extension of camera 80 cm.; $\times 72$.)

longer than those on the acute deficiency, showed necrosis of the epithelium, and a lack of inflammatory response. Typical changes are seen in Fig. 16 where necrosis of the epithelium is seen in a 117-day-old deficient rat. Enlargements of Malassez's rests were occasionally seen.



Fig. 16.—Necrosis of epithelium with proliferation down onto the cementum in a pantothenic acid deficient female rat 117 days of age. (Rat B5668, Spec. 8601, Pl. 8443.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 10; extension of camera 103 cm.; $\times 252$.)

SUMMARY

Rats placed on a purified diet deficient in pantothenic acid from the day of birth showed the following changes in the oral epithelium:

1. Early hyperkeratosis of enamel and mouth epithelium, noted in several rats as young as 21 to 48 days of age.
2. Necrosis of epithelium, first seen at 48 days, advancing deeper and appearing with greater frequency in older rats.
3. In several rats surviving more than 72 days, the necrotic process had advanced deeply, destroying all epithelial cells of the interdental papilla and the underlying connective tissue down to the crest of the alveolar bone.
4. Resorption of the alveolar crest had occurred in the advanced stages of tissue destruction in a few rats.

5. At all stages up to the complete destruction of the epithelium the vital cells of the basal layer had proliferated, thus protecting the underlying tissues from exposure to the oral cavity.

6. An unusual absence of inflammatory response was noted, notwithstanding the presence of marked destruction of tissue.

7. The changes in pantothenic acid deficiency differ markedly from those in dogs resulting from lack of niacin; which, although it is also a member of the B group of vitamins, results in a severe inflammatory response with hyperemia and edema.

Rats placed on the deficient diet from weaning showed:

1. The typical necrosis of epithelium, confirming the pathologic picture observed in the rats deficient from birth.

2. These changes occurred on superior basal diets high in either carbohydrate (64 per cent sucrose) or fat (48 per cent fat, 24 per cent sucrose) and in spite of deferring the start of the deficiency, thus enabling the animal to mature further.

3. Epithelial spurs from the surface and enlargements of Malassez's rests were observed.

The outstanding characteristic changes in pantothenic acid deficient rats, which confirm the earlier findings on the older filtrate fraction deficient dogs and emphasize the constancy of the pathologic changes, are: progressive necrosis of the oral epithelium and subsequent destruction of the underlying connective tissues with an extraordinary absence of inflammatory response.

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THE DEFICIENCY ANEMIAS

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DURING the past two decades much has been added to our knowledge of the pathologic physiology of anemia. The etiologies of certain types of anemia that heretofore had been unknown have been discovered. Furthermore, the development of newer laboratory techniques has given the physician means with which to make more exact differential diagnoses of the various anemias. Finally, the discovery of potent medication has given a more effective means of treating the anemias.

ANEMIA DUE TO DEFICIENCY OF THE ERYTHROCYTE MATURATION FACTOR

In the ten-year period, 1918 to 1928, three groups of investigators made extremely valuable contributions to the literature on anemia. The first of these was the report of Whipple and his associates¹² wherein it was disclosed that dogs which had been rendered anemic by frequent bleeding and a diet deficient in iron showed marked hemoglobin regeneration when fed whole raw liver. A year after this Minot and Murphy⁹ recorded their discovery of the efficacy of liver in the treatment of pernicious anemia in man. In 1928 the fundamental importance of the pathologic physiology of the gastrointestinal tract to the development of this type of anemia was elucidated by Castle and his associates.² The sum total of this work clearly demonstrates a relationship between food and gastric digestion. It is apparent that normal maturation of the red blood cells requires an erythrocyte maturation factor (E. M. F.) which probably is formed by the interaction of food and digestive juices in the stomach.

Addisonian pernicious anemia may be cited as an excellent example of anemia resulting from a deficiency of the erythrocyte maturation factor. This disorder is characterized by changes in the hematopoietic and central nervous systems. It is of most frequent occurrence in persons beyond middle age, but it may be found during the second decade of life. The onset of illness is insidious. Weakness, lassitude, progressive breathlessness and palpitation resulting in impaired powers of concentration finally bring the patient to the physician. Often the patient complains of soreness of the tongue and numbness and tingling of the fingers and toes. On physical examination he appears to be well nourished but pallid. About 75 per cent of all patients will show varying degrees of degeneration of the posterior and lateral columns of the central nervous system. There may be loss of the mucous membrane along the lateral margins of the tongue. The employment of various laboratory procedures will reveal an absence of hydrochloric acid and enzymes from the gastric secretion. There will be an anemia wherein the average size of the erythrocytes is greater than normal (macrocytosis) and the hemoglobin will be increased (hyperchromia) in proportion to the number of erythrocytes.

The significance of the achylia gastrica, which is always present in true Addisonian pernicious anemia, to the development of the anemia was demon-

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strated by the brilliant research of Castle and his associates. These investigators were able to demonstrate that patients with pernicious anemia, when fed beef protein alone, revealed no increase in the number of circulating red blood cells. However, when these same patients were fed a meal of beef protein predigested with normal gastric secretion each day, there followed a rise in the number of circulating erythrocytes which subsequently returned to a normal quotient. As a result of a series of investigations, Castle propounded the hypothesis that due to the gastric defect these patients lacked some substance (intrinsic factor) that was essential to elaborate from beef protein some element (extrinsic factor), the combination of which formed the erythrocyte maturation factor.

The *extrinsic factor* has been found in beef liver, kidneys, yeast, wheat germ, and to a less extent in eggs, milk, and tomatoes. It is heat stable, water soluble, and extractable from beef or yeast by 65 to 80 per cent alcohol.

The *intrinsic factor* in normal gastric secretion is heat labile as are many of the enzymes. It is destroyed by peptic digestion and is not pepsin, pepsinogen, rennin, or hydrochloric acid.

The *erythrocyte maturation factor* is found in liver, kidney, and brain. It is relatively heat stable and is soluble in water and in 70 per cent alcohol.

In the absence of the erythrocyte maturation factor, certain changes take place in the blood-forming tissues of the bone marrow. There is an increased cellularity of the bone marrow matrix. The precursors of the adult erythrocytes are more abundant than is normal, and among these may be recognized large numbers of erythroblasts and megaloblasts. This is to be interpreted as an inability of these cells to attain maturity and gives the appearance of reversion to an embryonic-type bone marrow.

Pernicious anemia is a distinct clinical entity, but any pathologic process that may prevent the formation of the erythrocyte maturation factor, its absorption, utilization, or storage may also lead to changes in the erythropoietic tissues similar to pernicious anemia. Thus, the removal of the stomach, cancer of the stomach, multiple adhesions about the intestines, sprue, and in some instances, pregnancy, and cirrhosis of the liver may be followed by the appearance of a macrocytic anemia.

Despite these valuable contributions there are many features of pernicious anemia that remain unexplained. Although the formation of an antipernicious anemia factor is dependent upon the interaction of an intrinsic factor of the stomach on beef protein, the defective physiologic process which leads to the development of achylia gastrica is unknown. Whether this be due to a hereditary defect, an avitaminosis, or even to some abnormality of the gastric nervous mechanism, is a subject that needs further investigation.

The possibility of there being a familial or hereditary predisposition to pernicious anemia has received more and more attention in recent years.

Meulengracht,⁸ in 1925, was among the first to present statistical evidence in favor of a hereditary factor in pernicious anemia. Schemm¹⁰ found five authenticated cases in the same generation. Furthermore, Schemm reported that in a study of 32 proved cases of pernicious anemia treated in his clinic there was a familial incidence of 18.7 per cent. Askey¹ reported the cases of 20 families wherein two or more members had pernicious anemia, making a total of 235 families which have been reported in the literature.³

CASE 1.—An instance of proved pernicious anemia which appeared in mother and daughter afforded an opportunity to make such a study in our clinic. The daughter, aged 28 years, was seen in the Out Patient Department in 1933. She complained of weakness,

palpitation, and dyspnea. Aside from obesity there was little to be found of clinical significance on physical examination. The important laboratory findings were achylia gastrica which persisted despite the parenteral injection of histamine; the red blood cell count was 2,000,000 cells per cubic millimeter; the hemoglobin content was 54 per cent of normal (7.5 grams Sahli); the halometer reading (average diameter of the erythrocytes) was 8.3 microns; the saturation index was 1.31. Accordingly, a diagnosis was rendered of hyperchromic macrocytic anemia.

Liver extract, potent for pernicious anemia,* was given daily by parenteral injection and was followed by a rise in the reticulocytes to 8.4 per cent of the total red cell count on the eighth day of treatment. On the twenty-fifth day of treatment the red blood cells were 4,100,000 cells per cubic millimeter, and the hemoglobin content was 88 per cent (12.4 grams Sahli).

CASE 2.—The mother, aged 60 years, also was examined in the Out Patient Department in 1933. She complained of easy fatigability and slight breathlessness on exertion. Physical examination revealed no abnormalities. An analysis of the gastric secretion revealed an absence of free hydrochloric acid. The red blood cell count was 4,170,000 per cubic millimeter. The hemoglobin content was 54 per cent of normal (7.5 grams Sahli). The mean corpuscular volume was 59 cubic microns and the mean corpuscular hemoglobin content was 18 micro micrograms. The anemia was classified as an idiopathic hypochromic and microcytic type.

The patient was given daily a solution by oral administration which contained 6 Gm. of iron and ammonium citrate (N.F.). This produced a slight increase in the reticulocytes above normal. On the fortieth day of medication the red blood cell count was 4,850,000 cells per cubic millimeter, and the hemoglobin content was 80 per cent (10.2 grams Sahli).

The mother did not report again to the Out Patient Department until February, 1943. On this occasion the patient complained of sore tongue of two weeks' duration. The red blood cells were found to be 2,700,000 per cubic millimeter. The hemoglobin content was 70 per cent (9.6 grams Sahli). The mean corpuscular hemoglobin content was 35 micro micrograms. The physical characteristics of the red blood cells, therefore, had become macrocytic and hyperchromic.

The patient received a parenteral injection of 120 units of liver extract during the first week. On the eighth day the reticulocytes were recorded as 7.2 per cent of the total red cell count. Thirty units of liver extract were given the patient at weekly intervals. Sixty days after treatment was instituted the red blood cell count was 4,420,000 per cubic millimeter, and the hemoglobin content was 90 per cent (13 grams Sahli).

Comment.—These two case records are cited to demonstrate the familial trend of pernicious anemia in a mother and her daughter. Of special interest is the occurrence of macrocytic anemia in the daughter at 28 years of age; this is unusual since pernicious anemia occurs most frequently in persons beyond the fifth decade of life. In addition to this it may be mentioned that the hypochromic anemia antedating the macrocytic anemia in the mother is also of infrequent occurrence. Both cases of anemia were undoubtedly of the Addisonian pernicious anemia type since there was a return of the red blood cells and hemoglobin to within normal range following the parenteral injection of liver extract.

Treatment of Macrocytic Anemia (Pernicious Anemia).—Potent liver extract is the most effective treatment for all types of macrocytic anemia. Liver extracts are now standardized in terms of the United States Pharmacopeia unit (U. S. P. unit) as defined by the committee on anemia in preference to the old method of assay expressed as "so much liver extract derived from so many grams of liver." One U. S. P. unit of liver extract is the daily amount necessary to produce a satisfactory reticulocyte response in ten days and a return of the hemoglobin and red blood cells to a normal range in sixty days in a patient with pernicious anemia who is in relapse. The ethical pharmaceutical companies have preparations available with concentrations of extract varying from $\frac{1}{2}$ to 15 units per cubic centimeter.

*Lederle's concentrated liver extract, 3 c.c. daily.

Parenteral injection of liver extract is the most effective means of treatment for a patient with pernicious anemia. A single injection of 45 units of liver extract may be given intramuscularly on two occasions in the first week. Following this, 15 units may be repeated once a week until the blood returns to normal. In an uncomplicated case of pernicious anemia in relapse the response to this therapy is dramatic. Within two or three days of the treatment the general appearance and well-being of the patient improves and there is an increase in appetite and strength. The glossitis gradually disappears in from one to two weeks.

On the third or fourth day young red blood cells (reticulocytes) appear in increased numbers in the circulating blood. These may reach a peak of from 15 to 40 per cent of the total red cell count on the eighth to tenth day, depending upon the severity of the anemia. A patient with a red blood cell count of one million per cubic millimeter should exhibit a reticulocytosis of 40 per cent if adequately treated and if there are no complications. A complete remission of the blood may be expected to occur within thirty to sixty days.

In patients whose illness is complicated by cystitis, prostatitis, cholecystitis, pneumonia, or some other infection, the response may be delayed. Under these circumstances much larger doses of liver extract may be required. Similarly, persons with neurologic involvement (posterior lateral column degeneration) will require larger and more frequent doses of liver extract. In early cases of the disease, less intensive treatment may be adequate. In well-advanced cases as much as 50 units biweekly may be required. Some physicians, influenced by the theory that a deficiency of some substance other than the erythrocyte maturation factor is responsible for the spinal cord changes, believe that the crude liver extract or thiamine chloride should be given in addition to potent liver extract. Subsequent treatment will vary in each case. An injection of 30 units of liver extract every three to four weeks is sufficient for the average case of pernicious anemia. One of the most important points, with regard to treatment, is the need to acquaint the patient with the nature of his illness and to emphasize that he continue treatment at designated intervals for the rest of his life. His failure to do this may result in relapse and disability resulting from neurologic changes.

ANEMIAS DUE TO IRON DEFICIENCY

Normally, iron plays an important role in one of the most important metabolic processes of the body, that is, the synthesis of hemoglobin. This process is a complex one and entails not only the need for adequate amounts of iron in the diet but its release from food, absorption from the intestine, distribution, storage, and utilization in the body. Furthermore, as a part of the body economy it must be retained and used repeatedly following the breaking up of the old red blood cells.

A decrease in the body's store of iron may lead to anemia. This may be observed in infants who have been fed milk over a long period of time without its being supplemented with iron-containing foods. Another frequent cause of this type of anemia is the gradual and prolonged loss of blood. Frequent and profuse menses, loss of blood from hemorrhoids, bleeding from peptic ulcer or other lesions of the gastrointestinal tract are frequently complicated by severe anemia. In pregnancy the fetus demands large amounts of iron from the mother so that subsequent pregnancies occurring soon after each other may deplete the mother to such an extent that she may become anemic.

Hemoglobin is the pigment-containing substance of the red blood cells. When it is decreased, the red cells show central pallor or achromia. The anemia, therefore, in contrast to pernicious anemia, is characterized by a proportionately greater reduction of hemoglobin than erythrocytes. Under these circumstances the color index is less than unity (per cent of hemoglobin \div red blood cells in millions \times 20), and when below 0.7 may be designated as hypochromic anemia.

There is one type of hypochromic anemia that may be found in women, especially during the period of puberty to menopause. In the medical literature this condition has various designations such as chronic idiopathic hypochromic anemia, simple achlorhydric anemia, and other less descriptive terms. Atrophy of the papillae of the tongue at the tip and along the border may be noted. The fingernails may be spoon-shaped (koilonychia) and brittle.

The almost constant finding of achlorhydria or hypochlorhydria in patients with idiopathic hypochromic anemia is now well known. Because of this observation, some studies⁷ were undertaken in our clinic to determine whether or not this abnormality could bear any relationship to the anemia. It was discovered that in the treatment of a group of patients who had achlorhydria and hypochromic anemia, the daily ingestion of a diet rich in food-iron produced satisfactory formation of hemoglobin only when the meal fed had been previously digested in vitro with hydrochloric acid and pepsin. It was interpreted that because of the deficiency of hydrochloric acid these patients were unable to get iron from food in sufficient quantities to maintain a normal hemoglobin saturation.

Heath, et al.,⁴ emphasized the importance of the menses as a contributing factor to this anemia. It has been estimated¹¹ that normally in the course of five years, a woman may lose as much as three liters of blood during her menses. Accordingly, increased or profuse menses may deplete the iron stores. Therefore, when iron requirements of the body cannot be met adequately, a state of chronic hypochromic anemia results.

When a diagnosis of anemia is made, a careful physical examination and laboratory tests should be performed to determine, if possible, some cause for the iron deficiency in the blood. There should be examination of the stool and urine at frequent intervals because of the possibility of occult bleeding. Observation of the gastrointestinal tract with the roentgen ray may aid in the discovery of some obscure or "silent" lesions. Among these might be mentioned stricture of the esophagus (Plummer-Vinson syndrome), diaphragmatic hernia, carcinomatous growths in the fundus of the stomach or the cecum, and hepatic flexure of the colon. Prolonged bleeding may occur from such benign lesions as a polyp in the ileum or ulceration of Meckel's diverticulum long before they produce any local signs or symptoms.

Treatment.—The treatment of anemia due to iron deficiency consists in the attempt to stop bleeding and the oral administration of iron in adequate doses. The cessation of hemorrhage in relatively mild cases may be followed soon afterward by a return of the hemoglobin to normal. In prolonged cases of bleeding the response to adequate doses of iron is very satisfactory. Ferrous sulfate, 5 grains (0.3 Gm.) three to four times daily after meals, usually will suffice to promote a maximal response of hemoglobin. Among other preparations of iron that will be found of equal value are Bland's pills, U. S. P., which should be prescribed in a daily dosage of 60 grains; iron and ammonium citrates, 90 grains; or reduced iron, 30 grains.

VITAMIN C DEFICIENCY

Vitamin C deficiency is recognized clinically by its hemorrhagic tendency. This tendency is due to weakness of the capillary walls caused by a lack of intracellular substance. Certain tissues are more prone to the hemorrhagic tendency than are others. Changes due to the increase in capillary fragility may appear first in the gums, which become swollen and painful. Petechial hemorrhages appear in the extremities and ecchymoses may appear following the slightest trauma. Hemorrhage and pain may be present in the joints. A careful analysis of the patient's dietary habits is most important. When it is learned that the patient has failed to ingest foods which contain vitamin C over a long period of time, the physician may well suspect the presence of scurvy. In addition, a plasma ascorbic acid level of 0.2 mg. per 100 c.c. is indicative of vitamin C deficiency.

Anemia is observed many times in persons with scurvy. Although anemia usually is normocytic, it may occasionally be macrocytic or hypochromic in type. There is much difference of opinion as to the cause of this anemia.⁵ Some investigators believe the loss of blood through hemorrhage is the main contributing factor, whereas others believe that vitamin C deficiency itself plays a major role by altering the bone marrow. Mettier, Minot, and Townsend⁶ demonstrated a reticulocyte response and the return of the blood to normal in patients with scurvy and anemia after they had been fed green vegetables and fruits. The anemia was not relieved by the intake of iron.

SUMMARY

This paper contains a review of the experimental work of the past twenty years which has contributed to the pathologic physiology of anemias. The fundamental work of Castle on pernicious anemia is reviewed and the treatment of this disease described. Although the etiology of pernicious anemia is unknown, some comments are made concerning a familial predisposition to this disease. As illustrative examples the case histories of a mother and her daughter are presented wherein it is shown that this disease was present in both. In addition, attention is called to the fact that hypochromic anemia had preceded the development of pernicious anemia in the mother. The role that iron plays in the development of deficiency anemia is also discussed. Although such anemia could result from loss of blood over long periods of time, it is stated that a similar anemia could occur in the absence of normal gastric secretion of hydrochloric acid. It is well known that anemia occurs frequently in persons suffering from vitamin C deficiency. Since this anemia is relieved by the ingestion of vitamin C, it is believed that an absence of this vitamin from the food contributes to the development of anemia.

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THE PHYSIOLOGIC ACTION AND BIOLOGIC EFFECTS OF THE B VITAMIN FRACTIONS

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BEFORE entering into a discussion of the importance of the B complex vitamins in dental medicine, we should have an understanding on a few important fundamental considerations.

1. It is false to assume that we are dealing with an entity, namely, the oral cavity. It is important to stress this point because many dentists (not all) by their actions and behavior, act as if the oral cavity is separate and distinct from other body tissues and has no relationship to the gastrointestinal tract, etc. In order to have an accurate understanding of the mechanism of the B vitamins and their place in dentistry, dentists must rid themselves completely of the notion that they are dealing with entities.

2. The accurate factual and scientifically provable assumptions which we must accept and on which we should act are that in biologic reactions we are dealing with dynamic, continuously changing multiple functioning process reactions, reversible in character when the environments are appropriate, but capable of alteration of function and ultimately of irreversibility if environments are unfavorable, which means that we are dealing with an-organism-as-a-whole-in-an-environment-as-a-whole; wherein events which happen, let us say, in the gastrointestinal system, the blood system, the endocrine system, or in the emotional environment, etc., can have desired or undesired consequences, let us say in the oral tissues.

Acting on these assumptions, then, we are dealing in this discussion particularly with the relationship of the B vitamins to the tissues of the oral structures.

Another important fundamental consideration is that the identity of any given vitamin is nothing more than a convenient label to describe certain, more or less, specific processes in the performance of which the vitamin plays a part. If we bear in mind two important things, namely, what a given vitamin does (its physiologic action), and the nature of the mechanism by which this physiologic action is accomplished, we proceed far toward understanding these important relationships in terms of biologic effects.

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The B complex is a mixture of many vitamins, the total number of which is not yet known. Of the known ones, at least nine are important in human nutrition as well as in the nutrition of animals, plants, and microorganisms. Others are known to be important in the nutrition of animals, plants, and microorganisms, but their importance in human nutrition has not yet been fully established. It is more than likely that they will eventually be shown to be important in human nutrition.

The B vitamins with which we are immediately concerned today, namely, those important to human nutrition, are B₁ or thiamine, B₂ or riboflavin, nicotinic acid, pyridoxine, pantothenic acid, choline, biotin, inositol, folic acid. In this discussion the principal stress will be laid on the phases intimately related to the clinical aspects, omitting references to their history, chemical nature, composition, structure, properties, etc.

GENERAL FUNDAMENTAL CONSIDERATIONS ABOUT VITAMIN FUNCTIONS

The primary physiologic action of the majority of the vitamins is not yet definitely known. Even in cases where we believe we have accurate knowledge (as in the case of B₁, B₂, and nicotinic acid), we do not know positively whether the reactions studied are primary or secondary reactions. Regardless of these limitations, there appear to be certain fundamental differences in physiologic action of the water-soluble vitamins and the fat-soluble vitamins. The fat-soluble vitamins (A, D, E, K) as well as C appear to function in maintaining the regulation of the metabolism of structural units. For example, vitamin A is fundamentally concerned with the building of cell nuclei. Vitamin E is concerned with the maturation and differentiation of cells. Vitamin D is concerned with the process of bone calcification. Vitamin K maintains the level of certain structural building units of blood. Vitamin C is concerned with the production of supporting tissues and the maintenance of intercellular substances.

The B vitamins on the other hand are concerned with the mechanisms of "energy transformation" or "energy metabolism." The mechanisms of energy metabolism involve consideration of a number of fundamental processes in the performance of which the B vitamins are intimately concerned, and without their intervention the energy metabolic processes cannot be completed.

An important aspect of energy metabolism, which is overlooked, but which must be comprehended if we are to evaluate B vitamin-tissue relationships, is that we must not act as if all there is to energy metabolism is the production or transformation of energy for what we may label "the performance of external work." An important part of energy metabolism is concerned with the transformation and production of the energy required for the performance of internal work within the cells themselves, and so energy transformation is of serious consequence as a factor in tissue integrity itself. Hence, a failure of energy metabolism can be intimately related to the actual production of tissue lesions, as we shall see in discussing the clinical aspects of the B vitamins.

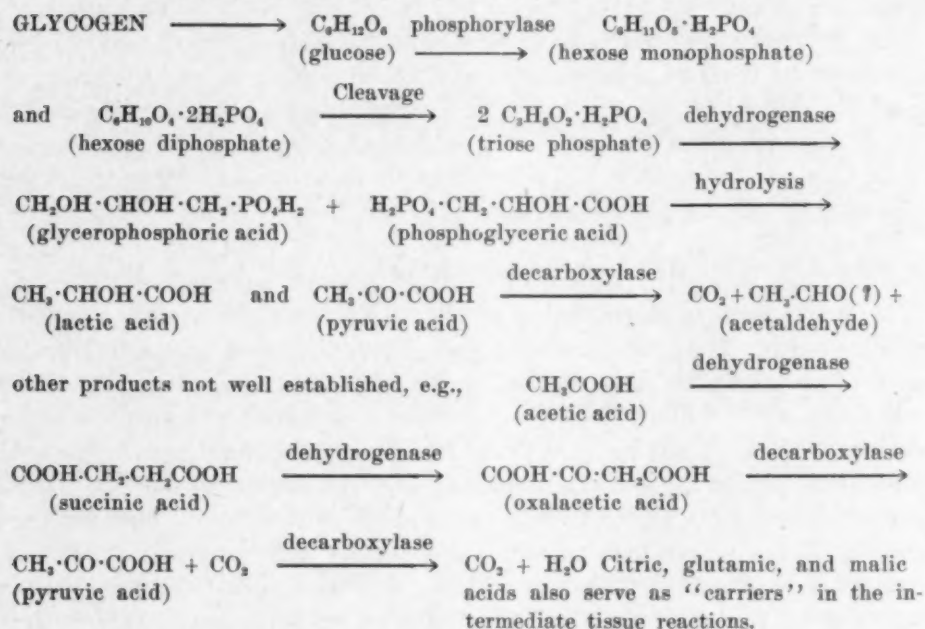
The transformation of carbohydrate (glucose, or blood sugar) in the cells of every tissue to ultimate energy is conditional upon the completion of a wide variety of reactions within the cells. Some of these reactions depend for their integrity of performance on the intervention and function of members of the B vitamins.

ENERGY (CARBOHYDRATE) METABOLISM

Before we can appreciate the physiologic action of the B vitamins, an understanding of the mechanisms involved in energy metabolism is necessary.

Molecules of glucose serve as nothing more than raw material in the first of many stages of intermediate, interrelated, step-by-step, energy metabolism, the ultimate production of energy being predicated upon the faithful performance of each and every intermediate stage.

Intermediate Stages of Carbohydrate Metabolism



If the sequential series of intermediate stages of energy metabolism are carried out, a happy result (production of energy) is achieved; the process is continuous and tissues function appropriately.

If there is failure to complete or maintain any one or several of these intermediate stages of carbohydrate metabolism, not only is there failure to produce the energy intended, but there is accumulation in the tissues of unwanted, undesired, intermediate products which have failed to be metabolized and which by their presence constitute cell irritants or cell toxins.

The mechanisms by which the intermediate stages of carbohydrate metabolism are performed and maintained comprise many systems of enzymes and coenzymes, some of which act on substrates consisting of the appropriate metabolites (intermediate degradation products) of the various stages; others of which procure and pass on the oxygen which is an essential ingredient of every type of cell reaction.

The enzyme systems which act on the substrates mentioned above are usually teams of enzymes—coenzymes which are specific in the sense that each enzyme performs a specific type of oxidation—dehydrogenation, decarboxylation, etc.—reaction for which another enzyme cannot substitute. The coenzyme part of the system usually has as a component part, an active chemical which we usually label a vitamin, e.g., the coenzyme, cocarboxylase is diphosphothiamine or thiamine pyrophosphate.

SECONDARY FUNCTIONS

We should bear in mind two other important aspects of function. We can have secondary function—the consequence of the primary function. Also, we can have incidental function due to an individual physicochemical characteristic.

For example, in its primary function, riboflavin never functions as riboflavin but in the form of a phosphate or dinucleotide. On the other hand, riboflavin itself happens to be photochemically active. Light converts riboflavin into a photochemically active compound which has a profound bearing on the stimulation of the optic nerve. Hence, riboflavin (*per se*) has an important incidental function in the visual mechanism of the retina.

PHYSIOLOGIC ACTION OF VITAMIN B₁

Vitamin B₁ in the form of diphosphothiamine or cocarboxylase functions as a component part of an enzyme system which controls at least one (but not all) of the intermediate stages of carbohydrate metabolism. The specific stage of which we have definite knowledge is one of the final stages, namely: the degradation (by decarboxylation) of pyruvic acid to acetaldehyde and carbon dioxide, or more likely to CO₂ and H₂O.

When the process of oxidative carboxylation of pyruvic acid takes place, happy consequences follow, energy is generated, and CO₂ and H₂O are eliminated by respiration. When the process fails to take place (lowered diphosphothiamine content of cells), oxidation of pyruvic acid at the desired rate cannot be maintained, cell pyruvic acid level rises, and nervous symptoms appear concurrently with symptoms of lack of energy. The development of nervous symptoms may be accurately described as the manifestations of a biochemical lesion, the first consequence of tissue depletion.

PHYSIOLOGIC ACTION OF VITAMIN B₂

Riboflavin takes part in a number of different enzyme systems which regulate cellular oxidation in tissues (respiratory enzyme systems). The apo-enzyme in riboflavin reactions is a specific protein. There are at least four different coenzymes containing riboflavin, a mononucleotide (riboflavin phosphate) concerned with carbohydrate metabolism and a dinucleotide (riboflavin-adenine-dinucleotide) concerned with amino acid metabolism, xanthine oxidase, and cytochrome reductase.

There is also evidence that riboflavin is connected with fat metabolism. An indirect function (through phosphorylation) of riboflavin is that it is involved in the absorption of glucose and galactose from the intestinal tract.

PHYSIOLOGIC ACTION OF NICOTINIC ACID

Nicotinic acid functions mainly as a part of enzyme systems which take part in protein as well as carbohydrate metabolism. At least two coenzymes contain nicotinic acid in the form of the amide, namely: codehydrogenase (I) which is diphosphopyridine nucleotide, and codehydrogenase (II) which is triphosphopyridine nucleotide. The mechanism of the enzyme action consists in the coenzyme absorbing two atoms of hydrogen from the substrate to form a dehydrocoenzyme which in turn is oxidized back to the original coenzyme (a typical reversible reduction-oxidation reaction).

PHYSIOLOGIC ACTION OF THE REMAINING B VITAMINS

Regarding the remaining B vitamins, little is known concerning their exact physiologic action, particularly the mechanism of their actions. Evidence is accumulating that some of these factors, such as pyridoxine, choline, etc., are concerned with fat metabolism while others are concerned with protein metabolism. In attempting to evaluate specific physiologic functions, we are at

present restricted to and guided by the known manifestations of their deficiencies in experimental animals.

PYRIDOXINE, VITAMIN B₆

Little is known regarding the physiologic action of pyridoxine. Evidence to date indicates that it is connected with the utilization of unsaturated fatty acids in fat metabolism and possibly concerned with protein metabolism.

PANTOTHENIC ACID

Little is known regarding the physiologic action of pantothenic acid. Its function appears to be related in some way to that of riboflavin.

BIOTIN, VITAMIN H

Physiologic action of biotin is not clearly understood. It is thought to be concerned with fat metabolism as a respiration enzyme. Biotin is also called coenzyme R, a respiration enzyme.

Its physiologic importance to date lies in the fact that it is rendered unavailable to the living organism by the formation of a complex with a protein constituent of raw egg white called "avidin." Avidin combines with biotin in the intestinal tract, preventing absorption of biotin and thereby producing biotin deficiency manifestations.

INOSITOL

Little is known concerning the physiologic action of inositol. It appears to act as a lipotropic factor. It increases peristalsis of the stomach and small intestines and may be a factor in gastrointestinal motility.

FOLIC ACID

Physiologic action is not yet known. Indications are that it is connected with the stimulation of growth of bacteria. Possibility of production in the intestinal tract by bacteria is also indicated.

CHOLINE

It is doubtful if choline should be classified as a member of the B group. It is both water soluble and alcohol soluble. It is included in the B group because it is usually found associated with the other B vitamins in sources of the B complex.

The importance of choline as a nutritional factor was a consequence of the discovery of insulin. As a result of the continuation of the work of Banting and Best, it was found that choline was necessary to prevent the development of fatty livers.

The physiologic action of choline is based on its lipotropic activity. The mechanism of its action is probably through its labile methyl group. Its major function is related to the mobilization of fatty acids in the body. In the absence of choline, the body is incapable of effecting the transformation of lipids to phospholipids with consequent accumulation of neutral fats in the liver.

BIOSYNTHESIS OF THE B VITAMINS

Some microorganisms are able to synthesize the B vitamins. Certain bacteria occurring in the gastrointestinal tracts of rats and in the rumen of cows

and sheep are able to synthesize B₁. Biosynthesis of nicotinic acid, pantothenic acid, inositol, folie acid, and biotin has been postulated.

Some of the B vitamins are essential for the growth of yeasts, microorganisms which in time stimulate the production of and in some cases synthesize additional vitamins. This growth-stimulating effect applies to pathogenic organisms as well as to beneficial organisms; for example, nicotinic acid is a growth factor for *Staphylococcus aureus* and for diphtheria bacillus. This property involves complications in connection with sulfonamide therapy indicating that the sulfonamides and the B vitamins are antagonistic. From another aspect, the B vitamins are essential to the growth of the intestinal flora. For example, pantothenic acid is essential for the growth of lactic acid bacteria. The use of sulfonamides inhibits the growth and development of intestinal flora, hence, following sulfonamide therapy there is not only a tissue depletion of the B vitamins, but also a marked diminution of normal intestinal and colon bacteria.

BIOLOGIC EFFECTS

Biologic effects can be expressed in two ways, either the beneficial consequences of appropriate physiologic function, or the undesired consequences of malfunction or dysfunction.

The consequences (beneficial) of appropriate physiologic action are usually expressed in terms of such labels as "health," "growth," "well-being," etc. The consequences (undesired, harmful) of more or less failure of function, we usually deal with more or less) are usually expressed in terms of such labels as "diseases," "clinical symptoms," etc. If we remember that we are dealing with the integrated cell metabolism of an organism-as-a-whole-in-an-environment-as-a-whole, we will refrain from identifying so-called "specific diseases" with specific deficiencies. Probably every clinical aspect of every so-called deficiency represents to some degree the composite picture of multiple superimposed deficiencies. This attitude towards clinical symptoms will go far towards explaining many apparent discrepancies and inconsistencies.

Referring back to the earlier paper, "The Fundamental Basis of Clinical Nutrition Applied to the Practice of Dental Medicine," it will be well to bear in mind the chronological sequence of development of clinical symptoms, namely, (1) tissue depletion followed by (2) biochemical lesions, followed by (3) altered function, followed by (4) anatomic lesions, and to remember that these conditions are detected clinically in the reverse order of their occurrence. Hence, in reviewing clinical aspects, if we realize that we are dealing with the consequences of events already happened in some respects and still happening in other respects, it will help in getting the "feel" of the clinical picture.

CLINICAL MANIFESTATIONS OF B VITAMIN DEFICIENCIES

The clinical manifestations or symptoms of vitamin deficiencies are nothing more than signs or indices of the consequences of disturbed function or malfunction or dysfunction resulting from inability of cells to carry out their various types of metabolism, either through lack of raw materials (substrates) such as amino acid, fat, carbohydrate molecules, or through lack of the enzyme systems—the activators which are necessary for the performance of the cell reactions. These clinical manifestations are usually found in every cell but are commonly detected and grouped as pertaining to specialized tissues.

It should be emphasized that in clinical practice we rarely, if ever, deal with a specific vitamin deficiency. Practically every manifestation listed under

B₁ deficiencies is to some degree or other attributable to other deficiencies such as deficiencies of B₂, nicotinic acid, amino acid, fatty acid, etc. Hence, in the following descriptions, the more accurate approach is to attribute the deficiencies predominantly but not entirely to lack of the vitamin in question.

VITAMIN B₁ DEFICIENCIES

Bearing in mind its biologic action, lack of B₁ undoubtedly brings about changes in every tissue of the body. The symptoms which have been detected and ascribed to B₁ deficiency are usually grouped as: (1) "Neurasthenic" manifestations. (2) Nervous system manifestations. (3) Circulatory system manifestations.

1. Under *neurasthenic manifestations* are included such complaints as fatigability, weakness, lack of energy, insomnia, aversion to food, headaches, irritability, poor memory, difficulty in concentration, together with gastrointestinal pains and disturbances, constipation, etc.

2. *Nervous system manifestations* include neuropathies of the lower extremities including burning sensations of the toes, soles of the feet, cramps in the calf muscles, loss of knee jerks, loss of vibratory and position sense. In more severe cases, manifestations include paralysis of eye muscles, stupor, failure of muscle coordination, various types of psychoses consisting of loss of memory for recent events, disorientation for time, place, and person.

3. *Circulatory system manifestations* include labored breathing on exertion, palpitation, excessively rapid heart action, precordial pain, dilated cervical veins, a palpable liver, edema and serious effusions occurring with or in the absence of congestive heart failure.

Oral Manifestations.—B₁ deficiency produces no specific oral manifestations; however, immediate relief of pain in dry socket and prevention of dry socket are said to be associated with B₁ therapy.

VITAMIN B₂ DEFICIENCIES

1. *Ocular Manifestations.*—Usually a vascularizing keratitis with photophobia, dimness of vision, severe infection of the vessels of the cornea and sclera, burning of the eyes, lacrimation, and in severe cases, opacities of the cornea.

2. *Oral Manifestations.*—Linear fissures in the angles of the mouth, a reddened, denuded appearance of the lower lip, glossitis consisting of a flattening of the papillae of the tongue, which becomes a purplish red in color.

3. *Skin Manifestations.*—Seborrheic accumulations in the folds of the skin, especially in the nasolabial folds, around the eyelids, on the ears, a sharkskin-like lesion on the nose.

NICOTINIC ACID

Many symptoms attributed to nicotinic acid deficiency (particularly many pellagra manifestations) are undoubtedly due to multiple vitamin deficiencies superimposed on mineral, protein, fat, and carbohydrate deficiencies and imbalances.

1. *Skin Manifestations.*—(a) Inflammatory lesions on exposed surfaces precipitated by exposure to sunlight. (b) Lesions caused by plugging of the sebaceous glands with dry, grayish, yellow material giving a sandpaper or sharkskinlike effect. (c) Hyperkeratosis with increased pigmentation over bony prominences. (d) Lesions around the anus and genitalia. (e) Lesions on the lips which are red and scaly and in the corners of the mouth as cracks and sores. (f) Lesions on the external portions of the eyes.

2. *Blood Manifestations.*—Certain types of anemia.

3. *Gastrointestinal Manifestations.*—Nausea, vomiting, diarrhea, distention of the abdomen, constipation.

4. *Oral Manifestations.*—A glossitis with burning sensation, scarlet redness, and swelling; all of the oral mucous membranes become reddened with a resultant stomatitis, gingivitis, and pharyngitis, and ulcers along the sides, tip, and undersurface of the tongue as well as on the buccal mucosa.

5. *Nervous System Manifestations.*—(a) Neurotic and neurasthenic symptoms similar to those described under B₁. (b) Central nervous system manifestations consisting of numerous types of psychoses, dementia, loss of memory, disorientation, confusion, excitement, depression, delirium, encephalopathies characterized by clouding of consciousness, cogwheel rigidities of the extremities, uncontrollable sucking and gasping reflexes.

PYRIDOXINE (B₆) DEFICIENCY

In experimental animals (rats) a deficiency of pyridoxine is associated with aerodynia (eruptions on hands and feet).

Specific symptoms due to pyridoxine deficiency are not yet definitely known in humans. Improvement following the administration of 50 to 100 mg. pyridoxine hydrochloride in muscle rigidity and muscle weakness, in some cases of paralysis agitans, has been reported. Improvement has also been noted in cases of pseudohypertrophic muscular dystrophy, myasthenia, and idiopathic epilepsy.

PANTOTHENIC ACID DEFICIENCY

In experimental animals, pantothenic acid deficiency is associated with a dermatitis in chicks, a lack of pigmentation in the hair of rats, and lack of hair (baldness) in mice.

ORAL MANIFESTATIONS OF NICOTINIC ACID, PANTOTHENIC ACID, AND UNKNOWNNS OF THE B COMPLEX

In dogs, Becks, Wainwright, and Morgan have reported well-defined oral manifestations in the mucosa and underlying alveolar bone, which may be summarized as follows:

Prolonged nicotinic acid deficiency produces marked gingival and paradental changes. Severe inflammation of the entire mucous membrane occurred with an involvement of the tooth-supporting osseous structure, accompanied by marginal atrophy resembling the inflammatory type of paradentosis in human beings.

In deficiency of the entire filtrate fraction, gingival inflammation was practically absent. Changes in the oral epithelium showed characteristics of degeneration. In many instances the surface epithelium was reduced to a fine line, accompanied by a rapid desquamation of the necrotic layers which showed the various stages of karyolysis, karyorrhexis, and pyknosis. In some areas the epithelial covering had completely disintegrated and left the paradental connective tissue and peridental fibers exposed to the various external influences of the oral cavity. The alveolar and supporting bone showed marked osteoporosis and progressive marginal atrophy—without marked inflammation—presenting a condition resembling the atrophic type of paradentosis in human beings.

Deficiency of nicotinic acid in addition to deficiency of the entire filtrate fraction produced additionally severe inflammation and edema.

BIOTIN (VITAMIN H) DEFICIENCY

Biotin deficiency has been noted in chicks, rats, rabbits, and monkeys. Since biotin can be synthesized by bacteria in the intestinal tract, it is usually possible to produce a biotin deficiency only by extensive feeding of raw, dried egg white. The manifestations of (induced) biotin deficiency in experimental animals are a generalized pruritic dermatitis, coupled with seborrhea, alopecia, poor growth, and a peculiar kangaroo-like posture.

Biotin deficiency in humans has been induced in a number of cases artificially by feeding 30 per cent of the total calories in the form of raw, dried egg white. In the early stages the subjects developed a fine, branny desquamative dermatitis. Later, mental symptoms similar to those of B₁ deficiency developed. Still later, during the seventh and eighth weeks of the test, the subjects showed pronounced graying in the pallor of the skin and mucous membranes. While the hemoglobin content of the blood was reduced, the pallor was out of all proportion to the blood picture. These symptoms disappeared rapidly on administration of a biotin concentrate.

CHOLINE

It is important to remember that choline deficiency symptoms are produced experimentally in animals when the rations are low in protein and high in fatty acids devoid of phospholipids. The deficiency manifestations on such diets consist of the accumulation of neutral fats in the livers (cirrhosis of the liver), and hemorrhagic degeneration of the kidneys in the animals involved. The addition of choline (or other lipotropic factor) reduces the fat content of the liver. No evidence is yet available as to the manifestations of choline deficiency in humans.

B VITAMINS

In the light of available knowledge concerning the physiologic action and biologic effects of the B vitamins, a number of factors stand out clearly:

1. The B vitamins are intimately concerned with the production and transformation of both the external and internal energy of the organism-as-a-whole. What we know about the carbohydrate-B vitamin phases of energy metabolism undoubtedly applies with equal appropriateness to the amino acid-B vitamin and lipid-B vitamin phases.

2. Since the growth, repair, and maintenance of tissues from the beginning to the end of life are conditional upon the continuous availability of adequate quantities of energy, it follows that tissue growth is just as dependent on the B vitamins as a whole as on the provision of suitable amounts of proteins, fats, carbohydrates, minerals, and the other vitamins.

3. In clinical practice, the judicious use of the B vitamins as a whole in the form of natural sources containing the lesser known fractions as well as the better known ones should be stressed, rather than the five major synthetic fractions: thiamine, riboflavin, nicotinic acid (amide), pyridoxine, and pantothenic acid.

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THE FUNDAMENTAL BASIS OF CLINICAL NUTRITION APPLIED TO THE PRACTICE OF DENTAL MEDICINE

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THE science of nutrition has made such amazing strides in recent years that we are apt to overlook a number of serious problems connected with the application of experimental findings to clinical practice.

We, who are concerned with clinical nutrition, should be constantly aware that we are dealing with problems of human nutrition rather than with the nutrition of subhuman animals. The nutritional progress of which we are justly proud has been confined to a large extent to the field of animal nutrition, whereas progress in the solution of human nutritional problems (clinical nutrition) has lagged seriously behind. This is a serious statement to make, and will undoubtedly be challenged by enthusiastic nutritionists who can point with justifiable pride (?) to such progress as the enrichment of bread, flour, and other food items, the stressing of the "basic seven," etc., as evidence of the progress made in the improvement of human nutrition.

Nevertheless, an unemotional, extensional appraisal of the nutritional status at the human level—whether gauged by selective service data^{3, 4, 7} or by dental caries incidence studies¹ or by other means such as dietary surveys⁶—reveals a widespread incidence of varying degrees of clinical malnutrition notwithstanding the progress made in animal nutrition.

If we investigate why there is such a time lag between the development of nutritional research in experimental animals and its ultimate application to clinical practice, we become aware of a profound fundamental difference between human nutrition and animal nutrition. Animals do not have labels for foods or nutritional experiences, whereas we humans ascribe an amazing (and conflicting) number of labels (accurate and inaccurate) not only to foods, but also to the functioning of foods and to nutritive processes. In other words, in dealing with human nutrition, foods, nutritive functions, nutritive processes, etc., have symbolic values which do not enter whatever into the picture in dealing with animal nutrition. The consequences of these symbolic values superimposed on the nutritive values have a profound effect on human nutrition.

This fundamental difference is ignored in dealing with human nutritional problems. It accounts in a large measure, not only for the time lag in application, but also for the failure of nutritionists to comprehend the fundamental nature of clinical nutrition, for the understanding of which neurolinguistic and neurosemantic reactions must be taken into account.²

Because of these symbolic aspects, a number of consequences follow:

a. Foods are evaluated by humans, not necessarily for their denotations of nutritive value, but for their favorable connotations of social, cultural, racial, religious, or other significance arbitrarily assigned to them. It so happens (tragically) that the consequences of evaluating foods according to symbolic values have led inevitably to widespread dietary deficiencies, even in favorable economic environments, where purchasing power permits a practically unlim-

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ited choice of food. In no field of clinical practice is this condition so vividly exemplified as in dentistry.^{1, 5} The symbolic aspects of food probably more than any other factor are responsible for the emphasis on the preponderance of refined sugar products in caries-producing diets. (Examples of symbolic values: "the staff of life," soft drinks, cake, ice cream, pie, cookies, candies, are symbols of rewards, treats, celebrations, social prestige, etc.). The same neurolinguistic and neurosemantic environments which make dietary deficiencies possible also conspire to render difficult the process of correcting them. At the level of animal nutrition, ration deficiencies when detected are usually corrected routinely (if desired) by the simple procedure of adding the missing nutrients to the ration. In human nutrition, the only comparable procedure would be to institutionalize everyone, and serve a balanced ration or a mash to everyone. Such a procedure would undoubtedly produce such states of rebellion and resentment (neurosemantic and neurolinguistic reactions) that the resultant emotional upsets would interfere with the efficient digestion, absorption, and utilization of the most "perfect" mash. The only practical mechanism we have found to date to correct dietary deficiencies is to educate individuals to an awareness of their existence and to do something about making the necessary changes in their food habits.

b. Because of the symbolism integrated into our neurosemantic and neurolinguistic environments, our (human) digestive, absorptive, and metabolic processes are profoundly influenced by the attitudes, judgments, beliefs, compulsions, etc., which govern our behavior. As common examples, we have "allergies," anxiety-tension states, neuroses, etc., while affect profoundly the efficiencies of digestion, absorption, and metabolism. This situation is further complicated by other environmental imbalances. In our (human) state of evolution—due to the mechanization of our times—we have changed from an environment of comparatively great physical activity and leisurely tempo of living to one of comparatively reduced physical activity accompanied by comparatively increased tempo of living. Our former state of high physical activity required a food intake of 3,000 to 6,000 calories, yielding nutrients (proteins, fats, carbohydrates, minerals, and vitamins) somewhat adjusted to the then needs of living. Our present state of low physical activity is accompanied by a reduced food intake (1,600 to 2,400 calories), yielding insufficient nutrients to meet the needs of modern living.

The combination of these two situations provides the unfavorable environments which constitute the biologic background of the clinical picture.

I. TISSUE DEPLETION

The first fundamental consequence of dietary deficiencies, or inefficient utilization of nutrients, or any environmental situation requiring the expenditure of nutrients at a rate greater than the rate of supply, is depletion of tissues or failure of nutrients to be made available for biologic reactions.

In biologic reactions, we are dealing with a multiplicity of process reactions, constantly changing, reversible in character, appropriate in function and purpose (growth, differentiation, energy producing, etc.) when environmental factors are favorable, but inappropriate (undesired), changed in character and ultimately altered in function when environmental factors are unfavorable.

Under conditions of favorable environmental relationship, nutrients of every description, enzymes, hormones, etc., not only are available, but also are able to reach the cells and biologic reactions are able to proceed appropriately.

II. BIOCHEMICAL LESIONS

If environmental relationships are disturbed (e.g., if the diet is deficient) essential nutrients fail to reach the tissues, and biologic reactions proceed unfavorably. Continued failure to supply nutrients will deplete the tissues below levels of supply needed for maintenance of reversible reactions. Performance of function is impaired with the consequence that instead of an occasional unfavorable imbalance of reactions, repeated unfavorable reactions take place. Conditions of altered cell characteristics are reached which may be labeled *biochemical lesions*. Accumulation of pyruvic acid in cells as a result of B₁ depletion may be regarded as a typical example.

III. ALTERED FUNCTION

If biochemical lesions are allowed to persist, cell metabolism is so disturbed, through persistent failure to maintain reversibility of appropriate reactions, that inappropriate reactions are superimposed on the lawful reactions, resulting, in time, in altered function. Examples of altered function of especial interest to dentists are the elaboration of lactic acid in the mouth, and the preponderance of osteoclastic reactions over osteoblastic reactions resulting in osteoporosities.

IV. ANATOMIC LESIONS

Eventually, persistent altered function, which amounts to severe environmental maladjustments, ends in irreversible reactions which may be labeled *anatomic lesions*, which we identify as the conventional "diseases."

A serious human difficulty is involved here because our recognition of these events follows the reverse order of their development. Tissue depletion (reversible and therefore easily correctable) occurs first but is rarely detectable because of lack of knowledge and techniques. Anatomic lesions occur last, yet are recognized first, presenting us with a difficult problem because they represent reactions which have already reached the irreversible stage, and therefore are not so susceptible to response.

Reversal of this order gives us the fundamental blueprint for the development of mechanisms to ensure tissue integrity (preventive medicine and dentistry).

Nutrition applied to clinical practice already is beginning to provide techniques which make it possible to detect and avoid the late stages of tissue depletion at a time when it is relatively simple to maintain appropriate reversibility of reactions and so—tissue integrity.

The technique of diet analysis reveals and verifies the existence of deficiencies in the diet. If the diet is deficient, it may be safely assumed that tissue depletion has already taken place. It may further be assumed that since—under our present system—physicians and dentists as a rule do not see patients until anatomic lesions are already present, we are also dealing with a very high incidence of more or less altered function. The question then arises what can we do, what practical steps can we take to anticipate and so avoid the inevitable trend towards anatomic lesions. The answer is that biologic changes must be detected in "patients" before the changes reach the stage of altered function. Under our present system of evaluation, the patient at this stage is labeled a healthy person and by definition is not yet a "patient."

An interesting situation here presents itself. It so happens that in dealing with dental caries (unlike the majority of other "diseases"), the time interval between the commencement of tissue depletion and eventual production of anatomic lesions is comparatively short. Also, we already have in dentistry in

the *Lactobacillus acidophilus* test surprisingly accurate techniques¹ for the detection of stage II, viz., the presence of biochemical lesions. Hence, we have already available one mechanism for the practical treatment of the problem of avoiding the development of stage IV (anatomic lesions), and so prevention of caries.

Regarding application to other "dental diseases," we have the techniques of diet analysis, roentgenography, tests for metabolic function, blood, fecal, gastric analyses, etc., to guide us in detecting changes at least at the level of altered function. In the absence of more accurate techniques for detecting tissue depletion and biochemical lesions, the maximum probability of our evaluations goes no further than arrestment (e.g., in paradentosis) rather than prevention.

At the present time, techniques for the rapid detection and measurement of tissue depletion are only beginning to be developed. According as they become available routinely, we shall have at our disposal the practical mechanisms for achieving the goal of prevention.

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PSYCHOSOMATIC MECHANISMS IN ORAL DISEASES

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THE search for pathogenic factors in dental and paradental disease has for some time extended beyond the defined limits of the oral cavity. Through dental medicine the roles played by variations in the external or internal environments of the total organism have come under profitable investigation. More recently there has been a tendency to explore those less tangible phases of human life known as "emotion" or "personality" and to speak, as does contemporary medicine, of *psychosomatic relationships* in the etiology of disease.

The term *Psychosomatic Dentistry* first appeared in print apparently in 1943 in an editorial comment which made a plea for further studies. Early in 1944, Edward Weiss, M.D., published an article with the title "Psychosomatic Aspects of Dentistry."¹² Over many years, however, there have been sporadic

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articles dealing with the role of emotional reactions in the production and treatment of oral diseases. Experienced dentists could not observe the destructive effects of clamping and grinding habits without realizing that mechanical stress and strain were but a reflection or consequence of less tangible stress and strain within the personality. A. G. Brodie,² Milton Leof,³ L. R. Johnson,⁴ and M. B. Walker¹¹ are outstanding among recent contributors to studies of motor habits and malocclusion in their relation to the personality, while Paul J. Boyens¹ and B. S. Frohman⁶ have made important contributions to the psychotherapy of these sources of oral pathology. It is the purpose of this article to discuss the nature of psychosomatic mechanisms and methods for their investigation in dental practice.

Psychosomatic medicine investigates the role of emotional factors in the causation of disease. It is a new term designed to imply the essential unity of "mental" and "physical" processes in human organisms and to designate the integration of psychiatric methods of investigation with those of the other medical sciences. While great stress is laid upon the role of neuropsychic processes in the production of disturbed bodily mechanisms, we must also bear in mind that lesions originating in tissues of the body themselves may affect the central nervous system and thereby cause disturbances of mood and those higher brain activities we call "thoughts," "ideas," "judgment," et cetera. For a painful pulpitis may produce irritability and inability to concentrate and may seriously disturb one's moods and social relationships. In the main, however, psychosomatic investigations show that our attitudes toward life can be among the many factors producing dental caries or paradentosis. The clinical facts which psychosomatic medicine studies were known to the fathers of medicine, but it has been left to the research methods of modern neurophysiology and psychiatry, particularly psychoanalysis, to investigate the mechanisms by which notions, beliefs, and other evaluations, which are the products of the interpreting mechanisms of the brain, may produce distempered moods and their physiologic consequences.

Sherrington, a great English experimental neurophysiologist, said, "Emotion moves us, hence the name." Emotional states are subjectively experienced as feelings, affects, or moods. An emotional state also involves expression through such physiologic mechanisms as the smooth and striated muscles of the body, the glands, or the body fluids. We are "moved to tears," "paralyzed by fear," "tense with expectancy," "breathless with excitement," and "gnash our teeth with rage"; we have "a raging headache," "a burning tongue," "a foul mouth;" we become "sick with fear," "griped" by someone's "gall," and "impotent with rage." These are but a few of the everyday physiologic expressions of strong emotion.

Emotional reactions have their origin in quite primitive biologic mechanisms concerned with food getting, with protection from dangers, and with reproduction of the species; in other words, they occur in those organismal reactions most closely related to survival of the individual and of the race—hunger and sex. In humans they become very complex, because in addition to being directly aroused by external threats to life (such as starvation, conquest by enemies, or deprivation of affection), they may be activated indirectly by symbols or cues which have come to stand for, or represent, such external situations. Subjective states of fear or anger, together with their somatic expression in the form of trembling, dilated pupils, tumultuous heart action, clenched teeth, dry mouth, and other visceral reactions, may be produced in response to a real en-

vironmental danger, or to the thought or memory or symbol of one. Memories, phantasies, the anticipation or misinterpretation of facts can all arouse as strong (and sometimes stronger!) emotional reactions as can actual happenings in the external world.

Due to certain neurophysiologic laws, our emotional reactions are established early in childhood and remain as more or less automatic habit patterns throughout life. Attitudes of aggressiveness, defensiveness, and the like are characteristic of individual personalities but individuals may or may not be aware of their attitudes. Often they are hidden or disguised from conscious awareness because, for various reasons, they are unacceptable to a person's pride or they clash with accepted social standards. Whether conscious or not, the results or expressions of attitudes may be apparent to the subject or his acquaintances as varieties of muscular, visceral, glandular, and biochemical disturbances. Not only are somatic states of health or disease produced by our attitudes, but the lives and characters of other people are affected by the behavior resulting from such attitudes.

The development of attitudes composing a given personality is a result of the adaptation of inborn biologic strivings to the customs and demands of social living. Inborn creature-urges concerning nutrition, protection, and reproduction must be channeled into socially acceptable modes of expression. They become conditional upon interpersonal relationships. Food is obtained through the medium of people who provide it; protection calls for alliances with people and involves some kind of payment for their services; sexual functions and care of the young involve the highest kind of cooperation between individuals in forms sanctioned by the political, economic, and religious institutions of a society.

The immediate satisfaction and/or the form of gratification of most urges must often be postponed or altered to conform to the permissions and prohibitions of community life, such as meal hours and "decency." The postponed satisfaction of an appetite or its complete frustration produces more or less strong neuromuscular and other tensions within the organism. The renunciation or the acceptance of less adequate substitutes for the originally desired source of gratification also produces physiologic alterations of function (and eventually structure), as when, for example, meat and other proteins are, for various reasons, renounced, and achlorhydria with its consequences may develop. Habits of eating are conditioned by or copied from the customs of a race or the particular practices of one's parents. Aggressiveness or submissiveness toward people are developed according to the customs of a society and the intrafamilial relationships of childhood. Sexual conduct is established not only by cultural tradition but through the personal experience of early life.

Personal security depends to a greater or lesser extent upon the modification of these strong impulses in the interests of community life. Since the postponement, alteration, or substitution of satisfactions sought under pressure from deep organismal needs lead to varying degrees of physiologic or even morphologic change, it is not surprising that conscious and unconscious trends towards the overcoming of such frustrations develop. What may not be obtained openly can sometimes be gained secretly or by circuitous pathways. These evasive tactics themselves lead to further tensions resulting from fears of discovery and retaliation, and from feelings of guilt. Such anxiety or guilt can produce further factual frustrations through self-imposed inhibitions or punishment.

Incompatibilities and conflicts between impulses may develop as when the need for protection clashes with a coexisting trend toward independence; or when protection and security are obtained by means of acquiring power over others, which might clash with a need to be loved. An infantile urge for food may be represented or expressed by a frantic acquisitiveness of money (as a means to purchase food); and the tension resulting from the struggle for money in a competitive-aggressive society, like our own, may so alter gastrointestinal and other functions as to prevent assimilation of the very food the neurotic adult so urgently sought, as in the fable of King Midas. Drives for sexual gratification not only clash with morality, but their aggressive aspects are incompatible with coexisting tender, protective, altruistic trends within the personality; the tension thus produced can lead to such alarming symptoms as frigidity, impotence, and other marital troubles.

Thus the expression of strong sexual impulses or the venting of rage may be met with such strong counterimpulses or prohibitions within a personality that the original desire is excluded from consciousness and only an awareness of anxious tension remains. Not only are we afraid to become angry or passionate but frequently we are afraid because we *are* angry or passionate. Our ideals and our conscience supervise and restrain us.

Anxiety is produced, then, when our impulses clash with the external realities of social existence or when they conflict with each other.⁷ Whatever the source of anxiety, physiologic tensions will be the consequence. Transient tensions are normal and part of our adaptive mechanisms, but chronic tensions arising from lifelong attitudes of defense and aggression lead to structural changes in those tissues or organs through which they are expressed. It has been estimated that 50 to 70 per cent of all human illnesses originate in this way.

Through the prolonged vascular congestion of tissues the soil is prepared for bacterial growth, or for degenerative fibrosis. The hypertenseness of musculature produces not only muscular aches and pains but fibrositis and even chronic arthritis. Posture is a gesture toward life. Continual stimulation of the emergency system of the body can produce high blood pressure, arteriosclerosis and, eventually, heart disease. Repeated disturbances of gastrointestinal motility and secretions lead to faulty assimilation so that metabolism may be dangerously affected despite high caloric, nutritionally correct diets. Stress falling on any one organ or system automatically involves others so that vicious circles are produced, as when bruxism leads to paradentosis, then through malnutrition to the failure of other organs and back to increased tooth grinding because of increased feelings of inferiority. Many links may develop in such vicious circles to be further complicated by infections, avitaminoses, degenerations, et cetera. Up to a point, psychosomatic reactions are reversible and more or less complete recovery can take place; but they may insidiously become irreversible and permanent structural damage result.

A considerable amount is known about the neurophysiology of anxiety and its somatic resonances. Certain neural pathways, the principal stations of which are found deep in the center of the brain in a region known as the diencephalon, mediate the consciousness or subjective awareness of affects and, also, their objective expression via the lower centers of the brain to the muscles and viscera of the body. A large mass of diencephalic tissue, called the thalamus, gives us awareness of strongly contrasting moods of pleasure or pain when stimulated by incoming nerve impulses from the various receptors or sensory endings of the body. A very small plate of tissue situated between and below

the thalamic nuclei contains groups of nerve cells which govern the rise and fall of temperature, variations of metabolism, cardiovascular and respiratory fluctuations, sleeping and waking, certain activities of the pituitary gland (with which this area is directly connected), and stimulate all visceral activities involved in emotional expression. This structure is known as the hypothalamus. It is somewhat analogous to an organ console containing many keys and stops—the various nuclei in the hypothalamus—which may be stimulated singly or in combination to produce the complex tones of emotional expression. Nerve pathways from the hypothalamus act on centers in the brain stem from which, in turn, impulses are relayed over the cranial nerves and down the spinal cord to the thoracic, abdominal, and pelvic viscera and to various glands and blood vessels.

These neural structures mediating emotional experience and emotional expression are very old and constitute one of the most primitive regions of our nervous system. They are surrounded by the cerebral cortex with its expansive convoluted plate of gray matter and central white association pathways. These cortical structures are the most recently acquired portions of the nervous system both phylogenetically and ontogenetically. One of the chief functions of the human cortex is to make abstractions from the information reaching it via sensory channels and to recombine those abstractions into new higher order abstractions which may in turn be represented by symbols such as pictures or words. These pictures or words become associated with thalamic feeling tone and thereby become the vectors of emotional experience. The cortex analyzes and refines emotional experience, giving us awareness of qualities and kinds of pleasurable and painful experiences blended with each other in ever new combinations. As far as emotional expression is concerned, cortical centers send impulses to the hypothalamus to produce emotional expressions consonant with the evaluations—be they delusional or correct-to-fact—that those cortical stations have elaborated.

While these psychosomatic mechanisms have been worked out in considerable detail for many diseases known to physicians and surgeons, little has been done in the field of dental medicine. Experimental observations have demonstrated that the pH, the viscosity of saliva, and to some extent the mineral constituents, may be altered under the influence of fear, rage, or pleasure. Calcium and other ionized salts of the body fluids are affected by emotional states as are the white and red cell counts of the blood.⁴ Excessive occlusal stress leads mechanically to paradentosis. Faulty mouth habits have been repeatedly demonstrated to be the expression of attitudes such as repressed rage or fearful dependency. It is obvious, of course, from what has been said before, that attitudes leading to inappropriate food habits, can lead to chemogenic sources of dental caries as when the excessive use of refined carbohydrates produces a florid acidophilus culture. Hypocalcemia and osteoclasia can result not only from attitudes involving incorrect food selection but from direct disturbances of the hypothalamus by anxiety despite an adequate intake of calcium.

THE CLINICAL INVESTIGATION OF PSYCHOSOMATIC RELATIONS

The practical application of psychosomatic principles in dental medicine (as in all other medical specialties) consists in the investigation of a patient's attitudes toward life. This is simply a matter of thorough history taking. Two points should be investigated in addition to the conventional inquiry about present, past, and familial illnesses.

The first is that of the *timing* or the sequence of symptoms and signs in *relation to* stresses and strains in the patient's social life (such as those occurring in connection with births, deaths, marriages within the family, and those occurring in the extrafamilial fields of friendship and business associations). A tactfully directed inquiry into the sequence of changing social relationships will usually show striking temporal correspondence with disease processes as, for example, when a gingivitis or a lichen planus appear and recur whenever certain patients pass through financial or marital stress, or when dentures are "accidentally" broken at times of business stress or frustrated affection. It is only necessary to list in three parallel columns the medical events, the social events and the year, month and day to see these relationships between disease and the personal life.

Stanley Cobb, in his small book, *The Borderlands of Psychiatry*,³ gives some excellent examples of this simple but effective method of history taking. Not only are pathogenetic factors from the emotional life of the patient revealed to the physician or dentist but a powerful psychotherapeutic tool is fashioned by means of which the patient may be given insight into his illnesses and incentive to change his ways of living.

The second point of investigation should be a study of the patient's "style of life," i.e., his emotional pattern or personality structure. This may often be inferred from the study of the timing of illness in relation to social events, or from the patient's attitudes toward the practitioner and his reactions to treatment, but the desired material is frequently elusive because subtle and, more often than not, disguised. Direct questioning is seldom helpful, for the patient either does not know himself or is afraid to reveal the nature of his deeper feelings. Consequently a sort of psychological sleuthing is necessary on the part of the practitioner who must put together the pieces of a jigsaw puzzle to find the design to which they belong.

This step of investigation often requires the services of a psychiatrically trained investigator—one skilled in interviewing techniques, such as a psychiatrist or psychiatric social worker.* What will be discovered by this phase of investigation is the type and intensity of the patient's emotional reactions, such as constant or recurrent anxiety, guilt, or rage, arising from basic personality trends such as passive dependency or active hostility. As B. S. Frohman⁶ has shown, the habitual clenching and grinding of teeth may express underlying emotional patterns of aggressive determination or gnashing rage. They may also express, symbolically, attitudes of stubbornness, masculinity, and fear of catastrophe. Thus, paradental diseases may be shown to have unconscious emotional drives as their fundamental causes.

Investigating the patient as a person in regard to the timing of his illnesses and his habitual emotional reactions reveals the indisputable fact that much oral disease is related causally to the emotional life. The relationship is frequently a reciprocal one. While a character trait such as rage—open or disguised—may result in occlusal trauma and consequent paradentosis, the disease process in turn may affect the personality if, for example, it is evaluated as humiliating, dangerous, or costly; a vicious circle may be established. A patient may literally "gnash his teeth at gnashing his teeth" in his rage at further interferences with his ambitious desires. Or his fear of oral disease and dental

*Psychological tests are of little value except to verify the impressions gained through the medium of one's own personality during person-to-person interviewing. The interviewing (history taking) is a therapeutic procedure itself inasmuch as the patient may gain valuable insights as he relates his story to a sympathetic listener.

treatment may maintain an anxious tension which will be reflected in his social dealings. The role of personal vanity in dental affairs is obvious.

The application of psychosomatic principles to dental medicine is a field wide open for exploration. As long as physicians, surgeons, and dentists limit themselves to their special treatments of specific organs or systems, they will never see the patient either as a total organism of interrelated organs and systems, or as a member of a community. The "oral cavity" is a verbal fiction as a moment's consideration shows. The region thus labeled is not only highly integrated with other systems of the body, but is the portal of nutrition and a principal organ of expression in social relationships. Not only should the integration of teeth and paradental structures with the total organism be considered, but the integration of the patient with other individuals in the competitive struggle for existence with its loves and hates, successes and failures, ought to be evaluated. Our attitude toward clinical problems should be less that of studying the disease in the patient, but rather the patient with a disease.¹⁰ Disease of any structure in the body to some degree involves neighboring and distant tissues; and to some degree it involves the patient's relationships with others, if indeed it did not originate in the disturbances of those relationships.

The one essential tool for exploration of this field is good history taking.⁵ Not only is a circumscribed disease process studied, but the sequence of events in the person's life leading up to and including the time of onset, such as states of worry, fatigue, frustration, et cetera, in business and family relationships, should be sought and thoroughly investigated. It will be amazing how frequent is the association of prolonged financial worry or marital incompatibility with dental caries or paradentosis. No formidable technique is required to make such investigations for such an approach is essentially a friendly, human one in which the physician or dentist interests himself in the patient's personal life, his feelings, his business and familial experiences, et cetera. It is only necessary to gain the patient's confidence and to penetrate his defensiveness. When the clinician expresses an interest in more than just a tooth or a liver in isolation, the relevant personal information usually pours out and the extra time required to be a "good listener" pays rich dividends in diagnosis and therapy. The patient's confidence is gained; he relaxes; he cooperates; health is more readily regained. Socrates, it is related, refused to prescribe a physic for Charmides' headache until he first eased his troubled conscience.

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COMPLEX ODONTOMA

REPORT OF CASE

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THE need for wider application and teaching of the fundamentals of dental medicine is especially apparent whenever a patient presents with a lesion slightly out of the ordinary. The dentist necessarily concentrates so much on repair and reconstruction that he welcomes the opportunity to add to his knowledge of the biologic processes involved in dental problems. He wants to review from time to time the less frequent pathologic lesions in order to maintain a constant awareness of these possibilities during his routine contact with patients. Such an obvious matter as the abnormal presence or absence of teeth is usually of great importance. In the following report a case is presented in which the clinical absence of one lower second molar was noted only because a complete examination was made at the time of treatment of two upper incisors which had been fractured in an automobile accident. The tumor discovered at this time was already so large that a young adolescent was in immediate danger of a pathologic fracture of the mandible, and, if untreated for a further period, of disfiguring destruction of the jaw.

Anamnesis.—A girl, 11½ years of age, was referred to the College clinic for the care of two broken upper incisors. As part of the dental examination, posterior bitewing roentgenograms were made. The roentgenogram of the left side, shown in Fig. 1, disclosed a large roentgenopaque mass distal to the lower first molar. This mass appeared to be extremely dense and was separated from the very thin superior cortical plate by a roentgenolucent area. Anteriorly the mass overlapped the distal root of the first molar. The upper second molar is seen in Fig. 1 to occlude with the gingiva below. The second premolar had not reached the line of occlusion and was impacted between the crowns of the adjacent teeth.

The patient had no complaint regarding the lower left second molar area and neither she nor her mother were aware of any abnormality. The upper central incisors had been broken in an automobile accident a month before but examination at the time revealed no other injury. There was no history of neoplasms of the jaws or skeleton in the family, and the patient had only suffered the ordinary childhood diseases and a slight "kidney infection" (at the age of 5 years). The patient's height was 161 cm.; weight, 51.8 kg.; blood pressure 120/80; pulse, 65; respiration, 14.

Status Presens.—This young adolescent appeared healthy and active with clear skin. Facial contours were symmetrical. Lymph nodes were not tender or palpable in the facial and cervical regions. Expansion of the mandible was slight but detectable by palpation in the region just anterior to the angle of the mandible on the buccal and inferior surfaces. There was no crepitation and the area was firm.

Intraoral examination revealed normal conditions except in the region of the lower left second molar. Although the other three second molars had already erupted, the lower left one had not appeared. Symptoms of expansion were evidenced by enlargement of this region of the mandible toward the buccal, lingual, and occlusal directions, by the imprint of the upper second molar on the surface of the opposing lower gum (which was not blanched and was of normal thickness), and by the impaction of the lower left second premolar. The area was nontender and no unusual sensations were to be found in the mouth.

From the Division of Dental Medicine, College of Dentistry, and the George Williams Hooper Foundation for Medical Research, University of California, San Francisco, Calif.

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Extraoral roentgenographic examination revealed a large roentgenopaque mass the size of a walnut in the region of the angle of the mandible. The lateral roentgenogram of Fig. 2 shows extension upward to the level of the first molar crown while distally the mass has expanded into the ramus in the direction of the mandibular notch. Anteriorly the mass has expanded past the distal root of the first molar, while downward it approaches the lower border of the mandible and has displaced the molar tooth to the point of interrupting the cortical plate. The mandibular canal is also seen to be severely displaced downward. The mass itself is a calcified structure of variable density with a mottled appearance but without cystic cavities. In some areas, such as the distal and the lower mesial portions, the calcification has occurred in lines. The mass is either attached to or closely approximates the crown of the depressed molar tooth. Completely surrounding the roentgenopaque mass is a roentgenolucent band varying in width from 2 to 6 mm. Bordering this is a narrow roentgenopaque wall of compact bone. The anteroposterior roentgenogram, Fig. 3, shows the extreme



Right

Left

Fig. 1.—Roentgenopaque mass distal to lower left first molar in a girl 11½ years old. Note absence of lower left second molar and impaction of lower left second premolar. (Pl. 8311.)

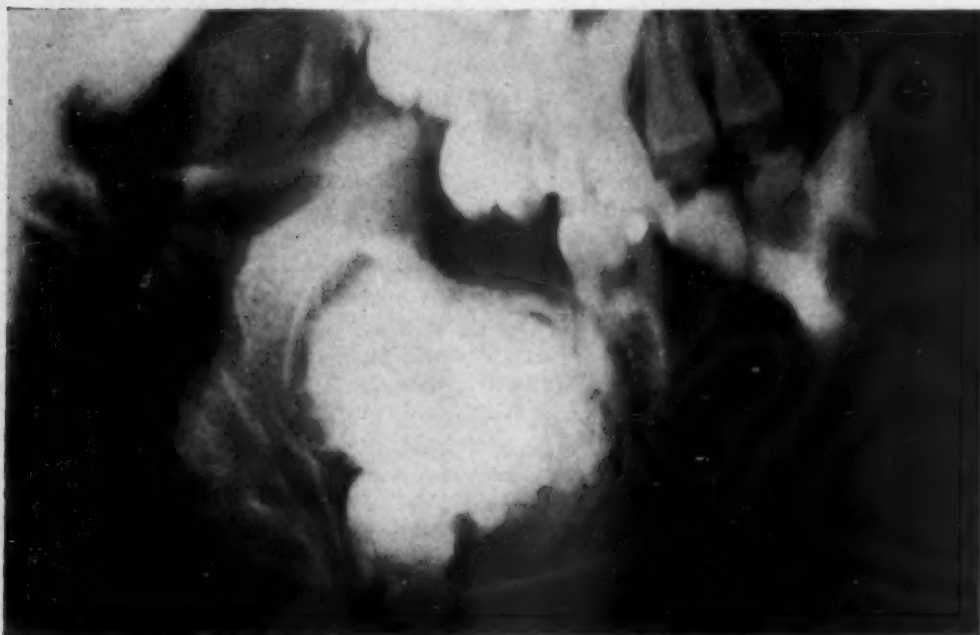
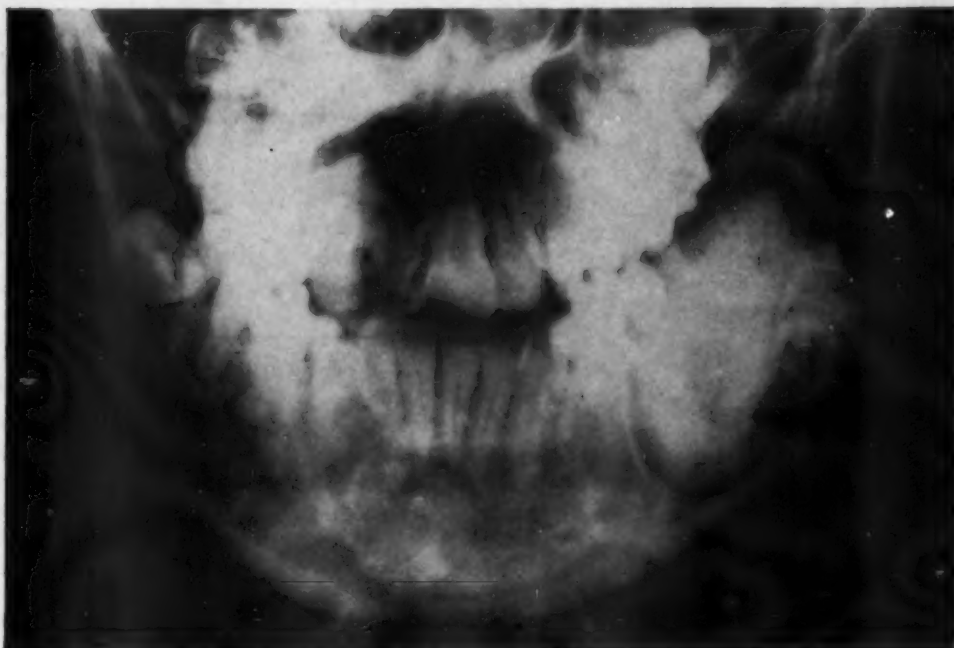


Fig. 2.—Roentgenogram of left mandible disclosing roentgenopaque tumor mass approximately 3 by 4 cm., which has depressed a molar tooth. There is no evidence of a third molar. Note the roentgenolucent space around the tumor and tooth, and the wall of cortical bone surrounding these structures. (Pl. 7803.)



Right

Left

Fig. 3.—Anteroposterior roentgenogram of the mandible showing buccal displacement of molar tooth (Pl. 7804).

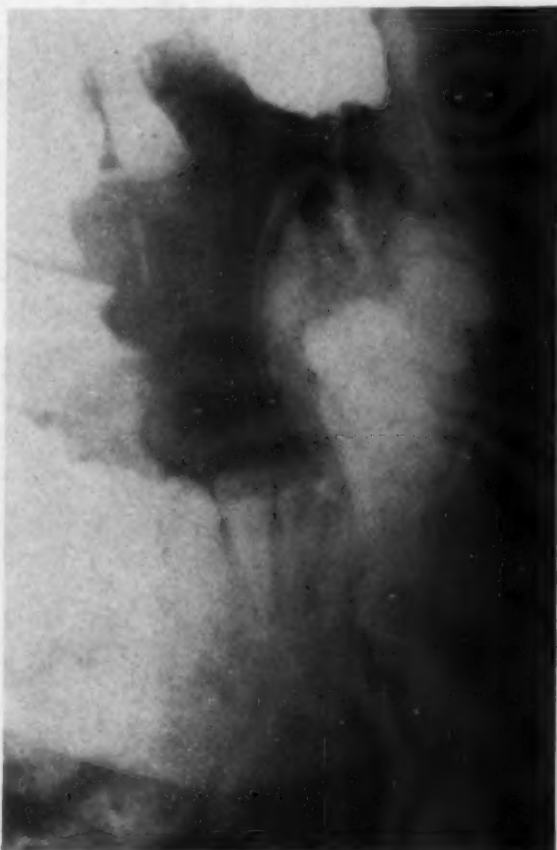


Fig. 4.—Roentgenogram of left mandible showing lingual expansion of tumor mass with exceedingly thin lingual bony plate (Pl. 8312).

displacement of the molar tooth in a downward and lateral direction. A roentgenogram taken from the occlusal direction, Fig. 4, shows clearly the extremely thin lingual bony plate and the surrounding roentgenolucent band. A lateral roentgenogram of the right mandible showed no abnormality. The lower right second molar was in occlusion and root formation was nearly complete. The lower right third molar was in its crypt and the development of the crown had reached the cemento-enamel junction.

Diagnosis.—The following factors suggest that this roentgenopaque mass is an odontoma: its location near the teeth in the mandible, its dense roentgenopaque nature with a mottled structure, absence of cystic cavities, the lack of formation of a third molar tooth on the affected side, the presence of a roentgenolucent band surrounding the mass, suggesting a capsule, and beyond this a roentgenopaque line which suggests a thin bony wall, and the age of the patient. These findings indicate a tumor with no tendency to invade surrounding tissues but which is growing and destroying neighboring structures by expansion. An osteoma is unlikely because of the location and character of the tumor and the lack of spongy structure. A cementoma is unlikely because the tumor is not associated with tooth-root structures and no history could be found of tooth extraction from this area. The absence of the lower left third molar would strongly suggest that an odontoma^{1, 2} had formed from its tooth bud and started to grow at an age early enough to expand widely before the second molar could erupt; thus by expansion the odontoma could bring about the observed depression of the molar tooth. That this is the second molar is further supported by the extent of root development, which already has passed the bifurcation and the apical third of the root is beginning to form. Due to the pressures from structures above and below it might well be slightly behind the right second molar in root formation. The right third molar, on the other hand, has only developed its crown, indicating that the depressed molar is ahead of it in growth. The mottled appearance of the tumor and its density make it possible that it is composed of dentine and enamel. The diagnosis suggested by this evidence is therefore a complex odontoma.²

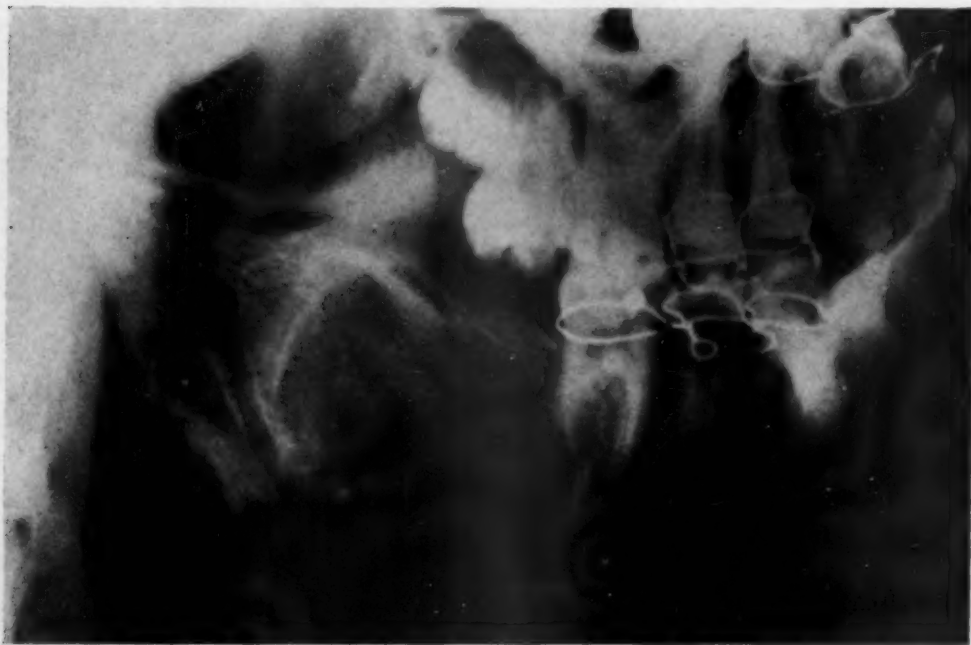


Fig. 5.—Postoperative roentgenogram of left mandible one week after removal of tumor mass and tooth. The teeth had been wired before operation and rubber bands were worn for five weeks beginning the day after operation. (Pl. 8313.)

Therapy.—Immediate surgical removal was indicated. Because of the growth of this tumor and of the extreme thinning of the bony plates surrounding it, there was danger of pathologic fracture of the mandible. An extraoral approach was indicated because of the size of the tumor, the many structures on the lingual aspect, and convenience of access.

Prognosis.—Aside from the possibility of surgical fracture the prognosis was very good.

Epicrisis.—The patient was hospitalized and the tumor excised by Dr. H. G. Bell, Associate Professor of Surgery, University of California Medical School. The teeth were wired

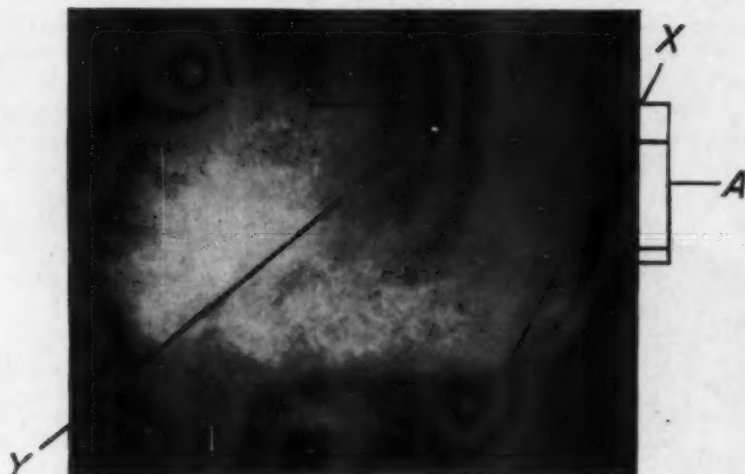


Fig. 6.—Roentgenogram of approximately half of the tumor mass. The other portions were removed in fragments. Note the density and irregularity of calcification. Several round structures resemble root cross sections (A). X and Y are the areas from which the sections of Figs. 8 and 10 were taken. (Pl. 8314.)

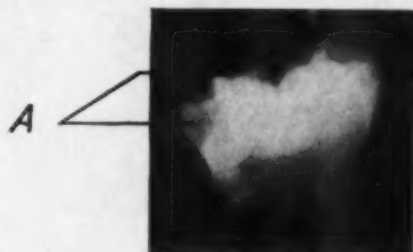


Fig. 7.—Roentgenogram of molar tooth depressed by the expanding tumor. Note enamel hypoplasia at A. (Pl. 8315.)

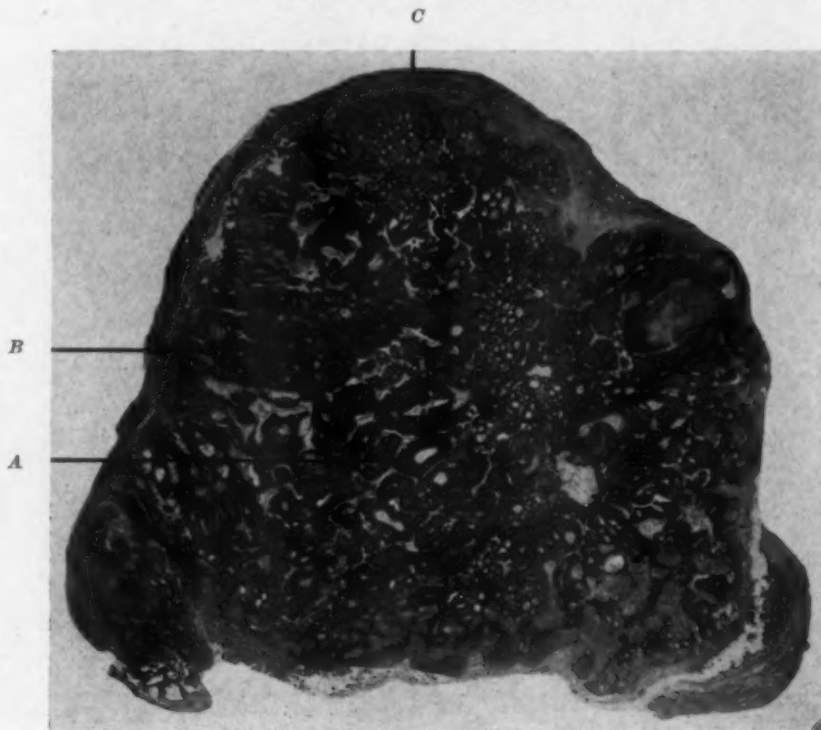


Fig. 8.—Photomicrograph of a large protuberance from the surface of the tumor at X, Fig. 6. Note the large number of rootlike cross sections throughout; in the center especially, dentine structures (A) with tubules and central soft tissue areas are seen. Amorphous calcifications appear at B. The entire tumor is enclosed within a capsule (C). (Pl. 8508.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Microtar f = 6 cm.; extension of camera 58 cm.; $\times 8.5$.)



Fig. 9.

Fig. 9.—Enlargement from the border of Fig. 8, showing fibrous capsule (A) enclosing the odontoma, and proliferating tumor cells (B) bordering amorphous calcified mass (C). (Pl. 8508.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; extension of camera 71.5 cm.; $\times 100$.)



Fig. 10.

Fig. 10.—Toothlike formation, from the center of the specimen, at Y, Fig. 8, typical of the complex odontoma. (Pl. 8510.) (Embedded in nitrocellulose, R.S. $\frac{1}{2}$ sec.; stained with hematoxylin eosin; Zeiss Microtar $f=6$ cm.; extension of camera 74 cm. $\times 11$.)

and prepared for intermaxillary elastics the day before operation as a precaution in the event of fracture. Following the usual premedication the operation was carried out under avertin and intratracheal nitrous oxide and oxygen anesthesia. The tooth and tumor were exposed without unusual result. The lower border of the mandible was interrupted by a soft area which proved to be next to the tips of the second molar roots. The buccal bony plate was only about 1 mm. thick. The tooth was removed easily although it seemed to be slightly engaged by the tumor. After removing half of the tumor from the lower and outward surfaces by chisel and mallet, the remainder was removed in one piece (3.5 by 2.5 by 2 cm.), with the adherent capsule. Some parts of the mass were like coarse sand. Surrounding the tumor there was a capsule of soft tissue which varied from 2 to 5 mm. in thickness. The cavity was found to be free of fragments; the soft tissues were closed over the wound with sutures and pressure dressing applied. Recovery was rapid and uneventful. Intermaxillary elastics were applied the day following operation for a period of five weeks. The patient was ambulatory the day after operation and was dismissed from the hospital on the third day. A lateral jaw film was taken one week postoperatively, Fig. 5, which showed a clean wound with loss of the lower buccal and inferior mandibular plates.



Fig. 11.—Magnification demonstrating typical enamel matrix (A), dentine with tubules (B) and amorphous calcifications (C) in decalcified specimen. (Pl. 8511.) (Embedded in nitrocellulose, R.S. $\frac{1}{4}$ sec.; stained with hematoxylin eosin; Zeiss Homal VI, apochromatic 20; extension of camera 71.5 cm.; $\times 345$.)

Röntgenograms of the tumor and second molar are shown in Figs. 6 and 7. Several circular structures resembling root cross sections are seen in the tumor. The mesial cusps of the depressed molar are deficiently calcified.

The tumor was cut into several slabs which were decalcified and prepared for *histologic examination*. Fig. 8 is a section through X of Fig. 6 and demonstrates numerous dentine structures which resemble root cross sections. Amorphous calcifications are also seen. A

capsule surrounds the tumor. Enlargement of the border, Fig. 9, shows a capsule, a layer of proliferating cells, and the amorphous calcifications.

In Fig. 10 is seen a section from the center of the tumor, at Y of Fig. 6, which discloses a toothlike formation. Such long rootlike structures are common in the complex odontoma. In this view the structure strongly resembles a tooth; cusps are seen at the top with a pulp chamber below; root canals extend downward with a large rootlike mass of dentine to the right. Further to the right the resemblance to tooth form ends as amorphous calcifications and enamel matrix are found.

Fig. 11 shows in higher magnification typical enamel matrix, tubular dentine, and an amorphous calcification.

Thus the tentative diagnosis is confirmed by the histopathologic findings: the tumor is a typical *complex odontoma*.

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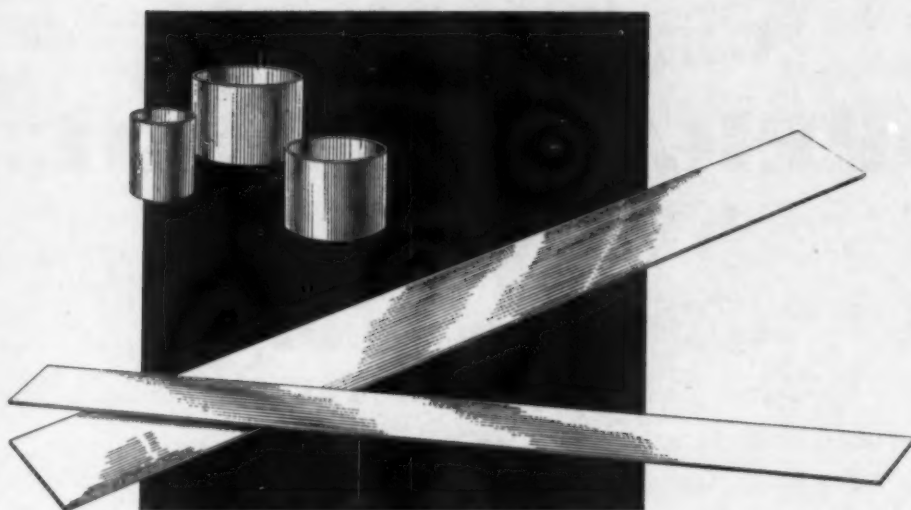
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Erratum

In the article by Dr. Sidney Sorrin, "The Use of Fixed and Removable Splints in the Practice of Periodontia," p. 354 of the June issue of the JOURNAL, all of the illustrations are credited to Miller: *Textbook of Periodontia*. This is in error. The credit line should be dropped from the following illustrations, which are Dr. Sorrin's originals: Figs. 5, 6, 7, 8, 9, 10, 11, 13, 14, 15, 16.

Announcement

In compliance with the directives imposed by the War Production Board limiting the amount of paper consumed in the production of this JOURNAL, the publishers find it necessary to change the format. As soon as these restrictions are lifted, the original format will be restored. Even though the number of pages has been reduced, the actual content of the JOURNAL has not been decreased to any appreciable extent.



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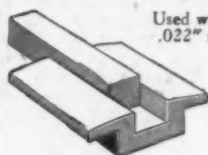
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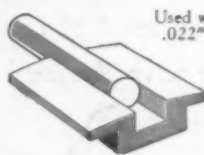
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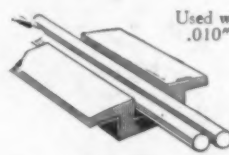
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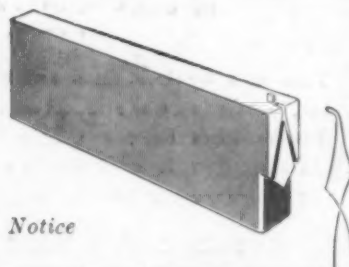
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AIDS TO ANESTHESIA

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INTRODUCTION

SINCE the field of anesthesia is pervaded with numerous difficulties and pitfalls, it is understandable that there are many men of the profession who frequently encounter trouble and who may even entertain a feeling of disgust or despair in regard to it. This article, therefore, is presented primarily for the purpose of offering some slightly different techniques and helpful hints in anesthesia in order to decrease the incidence of difficulty and to increase the efficiency of administration.

Especially recently, with our crowded offices, general anesthesia has been severely abused by rushing cases, and neglecting to apply the finer techniques which have helped general anesthesia reach the height of popularity it now enjoys. The knowledge of the more outstanding, important methods of general anesthesia is applied routinely, but, on the other hand, the numerous seemingly insignificant techniques are seldom used; when these are compiled and utilized together, they exhibit a marked improvement in the over-all anesthesia.

LOCAL ANESTHESIA

Our sympathy should go out to those patients who associate the pain of the needle with the anesthetic, thereby suffering the related mental agony of anticipation with the physical agony of the prick.

Due to the well-known fact that most of our patients fall in this category (not excepting the dentist and physician, but more so emphasizing them), our thoughts and procedures should be directed toward more considerate and less irritating techniques in the administration of local anesthesia.

For quite some time, even after the introduction of local anesthesia, the patient suffered excruciating pain when undergoing mandibular molar extractions because the operator knew nothing, at that time, of conduction anesthesia and, instead, used only the infiltration method. This technique, used alone, is no longer employed, of course, by the proficient operator.

While surveying a group of 500 patients, each of whom stated that the last dentist who had extracted a tooth for him was "good" or "not so good," it was found that these statements were made in reference to whether or not they were hurt during the operation.

There is a tendency with dentists, especially those receiving postgraduate training such as internships in charity hospitals and clinics, gradually to dismiss their sympathy toward the patient's sensitivity to pain. Thus, there is an inclination to rush the injection, to handle the patient in a rough manner, and

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to endeavor to satisfy him, at the same time, with "vocal" anesthesia. Certainly the origin of this is in our schools where the instructor in his effort to teach the approach of the needle to a definite anatomic position forgets to call to the mind of the student that the patient's feelings are inherently related to the site in question. If there is a disregard for sensitivity by the instructor, this attitude will be reflected in the student, causing the patient to exhibit greater fear on subsequent visits to the clinic. The patient should be handled carefully primarily, and the thought kept constantly in mind that human tissues are involved, and that the place to be injected is not a separate anatomic site independent of the patient's feelings.

It is the duty of each and every exodontist, when he has an operation under his surveillance, to know the techniques of eliminating pain and to be proficient in the administration of them.

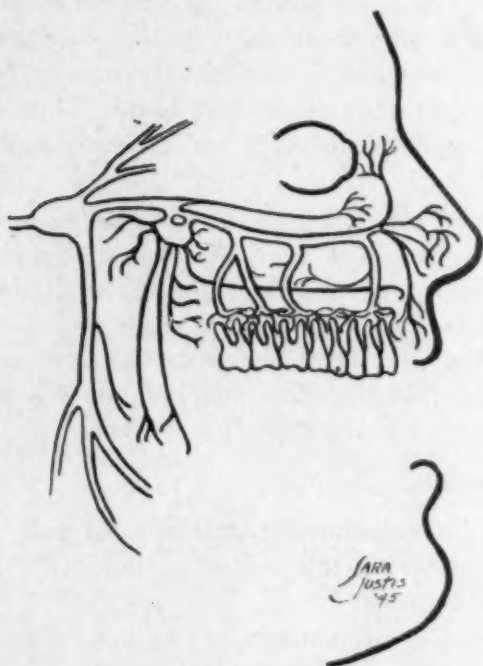


Fig. 1.



Fig. 2.

Fig. 1.—The distribution of the middle division of the fifth cranial nerve.

Fig. 2.—The *x* marks the point of entrance of the needle on the face when blocking the middle branch of the fifth cranial nerve from an extraoral approach.

POSTERIOR INFRAORBITAL (MAXILLARY DIVISION BLOCK)

It is commonly known that all of the maxillary teeth on one side of the arch with their surrounding tissues are supplied by several branches of nerves all of which are derived from a common branch beneath and behind the orbit. This branch is the second division of the fifth cranial nerve and is known as the superior maxillary or the middle division. It passes out of the skull through the foramen rotundum. There are two such nerves—one on each side of the midline of the skull. These nerves give the sensory supply to the entire upper dentition and the surrounding tissues. Therefore, it is readily obvious that by reaching these two nerves and depositing some local anesthetic solution, the necessary anesthesia is acquired for the extraction of any or all teeth in the upper arch. If this depth is not reached, several additional injections (approximately a total of nine) are required in order to anesthetize the patient suf-

ficiently for multiple extractions, including both sides of the upper arch. By giving this injection, the patient is spared the agony of anticipating further pain caused by the needle.

There are three ways in which this nerve may be reached at the desired location; one by the extraoral approach and the other two by the intraoral approach. One of the latter is preferred by this author. Usually the general surgeon or plastic surgeon will prefer the extraoral approach due to the theory of oral contamination.

The average dentist, in his training, has neglected the extraoral approach to all injections and, therefore, avoids them as much as possible. The technique for the extraoral approach to the maxillary division of the fifth nerve is as follows: The skin immediately inferior to the center of the zygomatic arch is cleansed with soap and water, then medicated with ether, merthiolate, metaphen, alcohol, or iodine. The needle is then inserted and carried medially, upward and slightly forward, until bone is reached; this will be at an approximate depth of 2 inches. One cubic centimeter of the solution is slowly injected and the needle withdrawn.



Fig. 3.

Fig. 3.—The intraoral approach to the middle branch of the fifth cranial nerve with the right-angle needle.



Fig. 4.

Fig. 4.—Intraoral approach to the middle branch of the fifth cranial nerve through the posterior palatine foramen.

One intraoral method in general use is accomplished with the aid of a right-angle needle. Using this technique, the needle is placed at the mucobuccal fold in the area of the third molar tooth and inserted upward, slightly backward and slightly toward the median line. The needle is inserted to a depth of 2 inches, measured from the crest of the alveolar ridge, at which time the point should be in close proximity with the trunk of the maxillary division anterior to the foramen rotundum. The above given intraoral method is most popularly used, but is less accurate than the other method which will be described in the following paragraph. The inaccuracy is due to the lack of landmarks or guidance while advancing the needle through the tissues.

The other intraoral technique is seldom used, nor is it known except by a relatively few dentists. However, it is very accurate and simple to administer. The technique is as follows: A $1\frac{1}{8}$ inch needle is adapted to a syringe with a short hub. The needle is then bent at the hub to form an angle of approximately 140 degrees with the syringe. With the bend or point of the needle upward, the posterior palatine foramen is located. Immediately after the mucous membrane is pierced, a few drops of solution are deposited. The needle is then advanced upward through this canal to its extreme length or until the hub has reached the mucous membrane of the palate. At this time the needle will be in close relation with the maxillary division of the fifth nerve anterior to the foramen rotundum. At all times during the advancement of the needle it is guided on its course by the bony walls of the canal which carries the anterior palatine nerves. There is no danger of getting out of the canal, thus the injection is rendered much more simple than any other method.

In either of the above methods, however, it is necessary to keep in mind the structures distal to the correct length of these needles.

MANDIBULAR NERVE BLOCK

Another injection which is often abused is the mandibular nerve block. Some of the essentials are often neglected by the instructors in teaching this procedure. Finally, in a great many cases, it has become a hit-and-miss action. Of course, it is for the most part satisfactory, because of infiltration. This happens in many cases, and the operator becomes impatient and gives a second and a third injection; each as haphazardly as the first. By this time the first has infiltrated and reached the nerve; therefore, it is presumed by the patient and operator that an unusual case is at hand, and the predicament necessitated three full carpules of the anesthetic solution. In reality half a carpule should suffice.

At present, most of our practitioners use the "swing" technique; that is, to insert the needle straight back then swing it over to the opposite side before further advancement is made. This is done in order to pass around the internal oblique ridge. This technique is very good for beginners if it has been correctly taught, for the landmarks are good and well defined. The failure with this method usually comes when the operator forgets to keep the syringe on a horizontal plane. Instead, he slopes it slightly downward in some cases, thus contacting the point of destination below the mandibular foramen. The direct thrust from the opposite side of the arch may result in the same failure.

Many warnings have been given, in the past, against injecting high for fear of missing the injection and inserting the needle into some vital structure instead. This, of course, is perfectly possible but seems to be no more dangerous in the hands of an average operator with average experience than any other method of anesthetizing the mandibular nerve.

An actual incident, related to me some time ago, often comes to my mind while teaching the mandibular injection. A clinician was presenting the technique of a third molar removal. It was noted, by some observers standing to the side of the patient, that the needle had extended from within the mouth all the way through the tissues and skin onto the outside of the face. The solution deposited at this time ran down the neck of the patient and was absorbed by his shirt collar. The story does not end here, for the strange part of it is that the operation was carried out to its completeness with apparently no pain to the patient.

What explanation can be made for this particular case? Was it psychological effect? I cannot believe it was. No explanation was given to me, but perhaps some solution was deposited when the needle passed the mandibular nerve, or maybe the mandibular nerve was actually stabbed, or the needle entered it injuring the particular fibers to the third molar tooth. Regardless of what happened, the procedure was a success and a freak which could probably never be intentionally repeated.

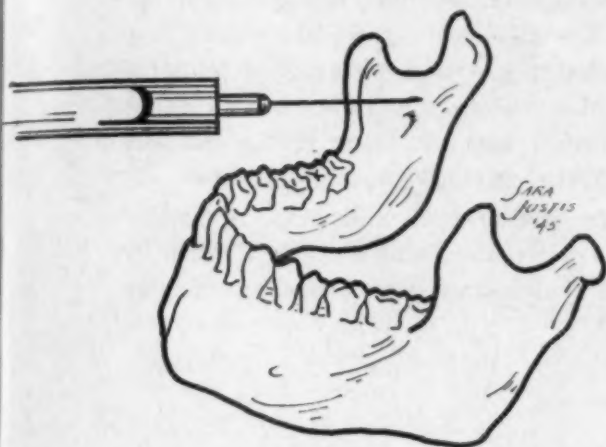


Fig. 5.

Fig. 5.—Position of the needle when giving the high mandibular injection.

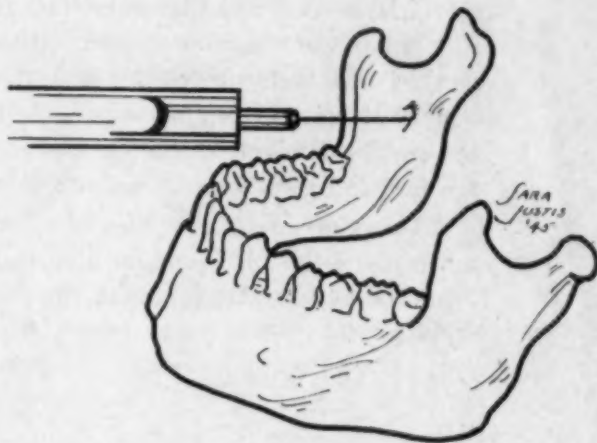


Fig. 6.

Fig. 6.—Position of the needle when giving the orthodox mandibular injection.

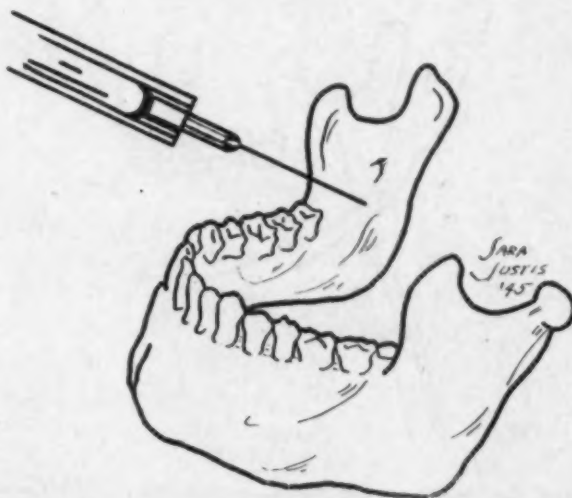


Fig. 7.—Position of the needle when the operator fails to hold it parallel with the occlusal surfaces of the teeth. This is often the cause of poor anesthesia.

A mandibular intraoral technique which I have found to be very helpful, and which has failed a minimum number of times, is that of a direct thrust posteriorly from a higher than usual level. This eliminates the swing, due to the fact that the needle is advanced above the internal oblique ridge. The technique is as follows: The palmar surface of the left index finger is placed on the mucous membrane of the cheek and the cheek is retracted. The tip of the index finger rests on the junction of the anterior border of the coronoid process with the anterior border of the ramus. In this position the finger is pointing on a line midway between the mandibular notch and the mandibular

foramen. The needle of the syringe, which is held in the right hand, bisects the fingernail on the left index finger. The needle is inserted into the tissues and carried posteriorly for approximately $1\frac{1}{4}$ inches, the needle remaining at all times immediately against the bone. At this point, 1 c.c. of the fluid is injected very slowly. Using this method, both the lingual and mandibular nerves will be blocked, with injury to neither. Anesthesia sufficient for operating purposes should be effected within three minutes.

I have used this higher method for some time, and have found it to be far superior to the regular standard method. The superiority of the above-described method has been proved to my satisfaction many times by giving bilateral mandibular blocks; on one side using the high injection, and on the other using the standard injection. Even though, in most cases, the latter of the two was the first injection to be given, it always effected operative anesthesia last.

In cases where abscessed teeth are to be extracted, anesthesia is usually much more difficult to obtain, due to the lowered threshold of pain. It will be found that the higher injection effects much better anesthesia in these particular incidences.

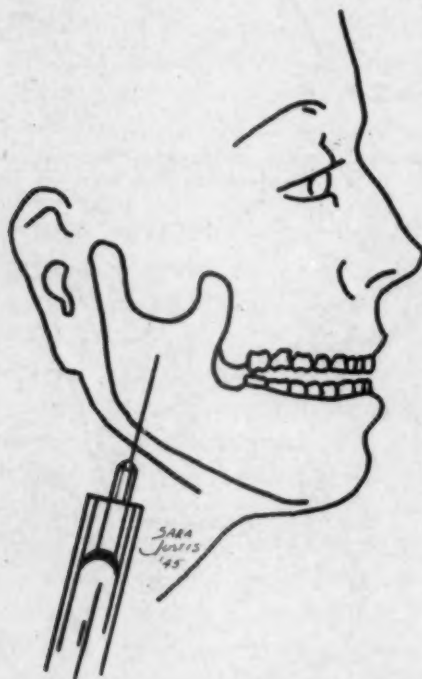


Fig. 8.—The position of the needle on the medial surface of the ramus when approaching the mandibular nerve extraorally from the angle of the mandible.

A SIMPLE EXTRAORAL TECHNIQUE FOR THE MANDIBULAR NERVE BLOCK

Occasionally, it is found necessary to obtain mandibular local anesthesia in a condition of trismus, ankylosis, or severe oral infection. In these cases the mandibular nerve is not accessible from an intraoral approach. An extraoral approach must then be sought. The most simple of these takes entrance along the lower border of the mandible at the angle. The technique is as follows:

The skin is first cleansed and premedicated at the angle of the mandible. The needle point is placed at the junction of the angle and posterior border of the ramus of the mandible on the immediate lingual or medial surface. The point is aimed in the direction of the mandibular notch and inserted to the

length of a $1\frac{5}{8}$ inch needle with a short hub. The needle should be kept in close proximity with bone at all times. At this point the fluid is deposited slowly and the needle withdrawn. There is a greater amount of pain involved with this injection as compared with the intraoral injection, and were it not for this, it could be considered superior and it is practically as simple.

ANESTHESIA FOR CAVITY PREPARATION ON MANDIBULAR TEETH

It is an undeniable fact that attempting to gain anesthesia for cavity preparation on mandibular teeth is about the most trying procedure imaginable in some cases. It should always be remembered that on the lower teeth a block should be used for this procedure, and not infiltration, regardless of the tooth involved. It is suggested by this author that a mandibular block be used for the molars and a mental nerve block for the premolars and anterior teeth. The foramen should actually be entered while giving the mental block in order to attain profound anesthesia.

The above-mentioned is often done with the result that there is still incomplete anesthesia. If, at this time, anesthesia is not complete, a lingual injection should be given as an infiltration with the point of the needle lying directly in contact with the bone as near the area of the apex of the root of the tooth in question as possible. At this point, about $\frac{1}{4}$ c.c. of solution is deposited and the needle withdrawn.

At all times, during the insertion of the needle, the operator should be certain to keep near to the bone in order to avoid the submaxillary or sublingual glands.

If this technique is followed closely, few mandibular cavities should be prepared without complete anesthesia.

THE MENTAL NERVE BLOCK

This particular procedure is so abused that seldom is it ever effective with the average general practitioner of dentistry. In some manner or other, a supremely distorted idea has been conceived by our fellow workers that there is no necessity of entering the mental foramen with the needle in order to obtain an active block, but that it is necessary only to deposit the anesthetic solution somewhere in its vicinity, and then, by soft continuous massage, the block will be effected. Due to the failures while using this technique, many have misplaced their faith in the mental nerve block. This is very bad, for actually it is one of the most profound blocks known to us when administered correctly. The foramen must be entered; then, due to the confinement of the mandibular canal, the nerve is bathed to the utmost even when small amounts of solution are used.

LOCATION OF THE MENTAL FORAMEN

Actually, the mental foramen is not difficult to locate, but due to the erroneous idea that it is hidden away, the injection is often avoided. In case of the mandible with teeth, the mental foramen lies midway between the alveolar crest and the lower border of the jaw superio-inferiorly and usually between the roots of the first and second premolars mesiodistally. However, this mesiodistal position often varies and may lie anywhere from slightly anterior to the first premolar root tip to slightly posterior to the second premolar root tip. When the jaw is edentulous, the foramen will usually, of course, be found closer to the

superior border than to the inferior—this is due to the great amount of resorption of the alveolar process which once held the teeth. The foramen, in most cases, may be located by palpation extraorally.

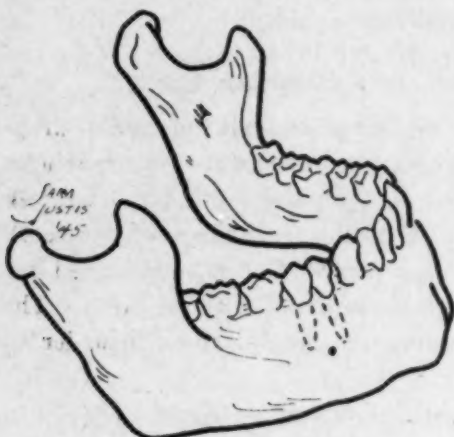


Fig. 9.

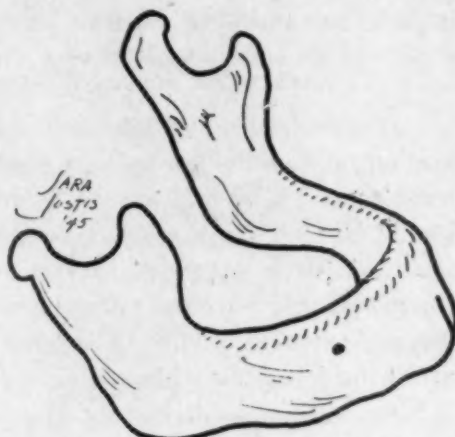


Fig. 10.

Fig. 9.—The position of the mental foramen.

Fig. 10.—The position of the mental foramen in the edentulous mandible, showing the opening to be closer to the upper border due to resorption of the alveolar ridge.

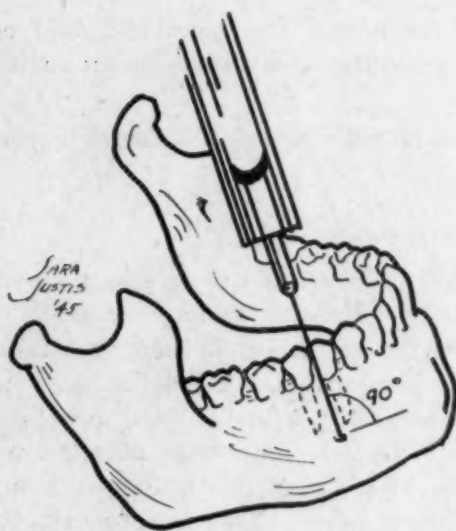


Fig. 11.

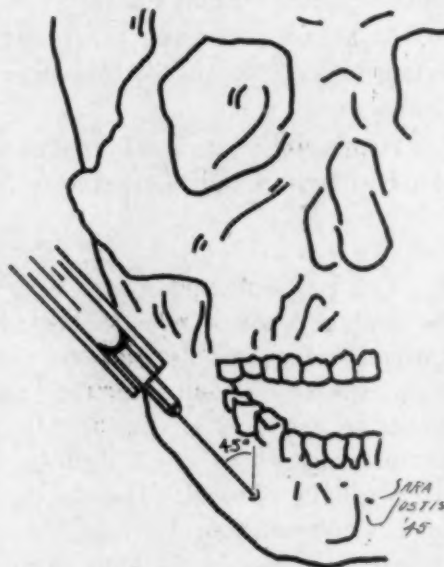


Fig. 12.

Fig. 11.—Lateral view of the needle in position while giving the mental injection.

Fig. 12.—Anterior view of the needle in position while giving the mental injection.

A SIMPLE TECHNIQUE FOR THE MENTAL INJECTION

When the right side is being injected, the operator may stand behind the patient with the left thumb retracting the cheek. The needle is inserted at the fold and advanced directly to the bone to the approximated position of the foramen. At this point, a few drops of the solution should be deposited so as to gain surface anesthesia in order to locate the foramen without pain. After a period of thirty to forty seconds, the needle point may be maneuvered and the foramen located. The needle is held at a right angle with the mandible anteroposteriorly and at a 45-degree angle with the mandible superio-inferiorly.

When the foramen is located, it should be definitely entered and from $\frac{1}{2}$ to 1 c.c. of solution deposited. When this is completed, the lingual soft tissues must not be forgotten. The needle should be inserted lingually to the bone at the approximate location of the apex of the root of the second premolar. At this position $\frac{1}{4}$ c.c. of solution should be deposited. Care must be taken to remain close to the bone with this lingual injection, so as not to enter the submaxillary gland.



Fig. 13.—Position of the fingers to locate the mental foramen extraorally.

A SIMPLE EXTRAORAL PROCEDURE FOR INJECTING THE MENTAL NERVE

The intraoral procedure very often is frowned upon by our plastic surgeons. Also, when severe mouth infection exists and anesthesia is necessary in this area, the extraoral technique is in order. Therefore, this procedure should not be feared but added to the mastered work of the operation. It, too, as found with the extraoral injection of the mandibular nerve, is more painful. The technique is as follows: The area involved should be cleansed and medicated on the surface. The mandible, inferosuperiorly not including the teeth, should be held between the index finger and thumb through the cheek. The needle is then inserted from the same angle as given for the intraoral method directly to the bone in the approximated area of the foramen. At this point, a few drops of solution are deposited. The foramen may then be located painlessly by repositioning the needle. After entering the foramen, approximately $\frac{1}{2}$ to 1 c.c. of solution is injected and the needle withdrawn. Here, also, the lingual injection must not be overlooked.

If either of the above mental nerve injections is followed out correctly bilaterally, surgery may be painlessly performed on any part of the mandible or mandibular teeth from the second premolar on one side to the same area of the opposite side.

ABUSING THE INFRAORBITAL AREA

Unlike the mental nerve block, the infraorbital canal does not have to be entered in order to effect a block of the infraorbital nerve unless there is an attempt here to block all teeth of that particular side with one injection. This most often fails. Due to the fact that many operators persist in doing this, there are many patients who are greatly abused. One case of blindness in one eye due to some operator's needless entrance deep into the infraorbital canal came under my observation. The most common disfigurement is the "black eye." This is actually a hematoma or a release of blood into the tissues, due to the piercing of the infraorbital vessels. This accident often happens and may be quite embarrassing to both the operator and the patient.

This injection, likewise, may be given either intraorally or extraorally, and both techniques are very simple. The operator need not endeavor to get closer than approximately 2 or 3 mm. to the opening of the foramen.

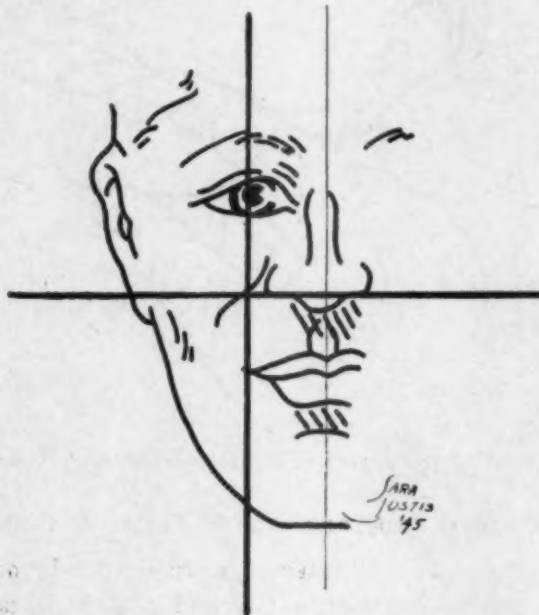


Fig. 14.—The position of the infraorbital foramen.

THE INTRAORAL APPROACH TO THE INFRAORBITAL NERVE BLOCK

The index finger of the left hand is placed over the foramen extraorally, the foramen having been previously palpated, then the upper lip is raised or retracted by the thumb. The needle is then inserted into the mucobuccal fold over the cuspid or first premolar tooth and advanced to the vicinity of the infraorbital foramen. This foramen is located at the intersection of a line passing through the center of the orbit with another line passing through the wing of the nose. This may be definitely done by palpating with the index finger. At this point, 1 c.c. of the solution is deposited and the needle is withdrawn. Massaging is not necessary.

THE EXTRAORAL APPROACH TO THE INFRAORBITAL NERVE BLOCK

This is, perhaps, the most simple procedure of any nerve block. The opening may be easily palpated with the index finger immediately under the center of the infraorbital ridge. The needle, held at a right angle to the surface of the

face, is then introduced through the previously cleaned and medicated skin to the vicinity of the opening, and from $\frac{1}{2}$ to 1 c.c. of solution is deposited. Mention should be made there, also, that a greater amount of pain is associated with the extraoral method, due to the toughness of the skin which must be pierced in order to reach the nerve.

In the opinion of this writer, the infraorbital nerve block is seldom ever necessary; but, instead, a high infiltration in this area will generally suffice.

ANESTHETIC SOLUTION CONCENTRATIONS

Concentration of the anesthetic solution and their vasoconstrictors seems to be the basis of considerable argument. These discussions concern both the immediate and the delayed reactions on the patient. The trouble probably is that the operator, in some cases, does not realize that when a stronger solution is used, it may be used in smaller quantities. It must be remembered that when the injection is correctly administered, it is not the amount of solution given that affects the depth of anesthesia, but the potency of the anesthetic. Sometimes it is necessary to use stronger solutions because the weaker will not carry the depth of anesthesia needed.

There must be a realization that an anesthetic solution given locally does not competely anesthetize the nerve, but only raises the threshold of pain to a certain degree. In the case of acute infection the threshold of pain is very low and difficult to raise because the nerve fibers are extremely sensitive.

It may be more easily understood if we compare the threshold of pain to an overflow pipe and the pain to water rising to different levels of this pipe. In this case, we shall say, there is no pain felt by the patient until there is a rise high enough to enter the overflow pipe. In further explanation, we might say, the overflow pipe is very short in incidences of infection; therefore, only a slight stimulus is needed to cause an overflow giving pain.

Now, for example, let us use our anesthetic solution in three concentrations, as "weak," being compared to a 1 per cent procaine, "medium" as compared with a 2 per cent procaine, and "strong" compared with 4 per cent procaine. Then, we might easily see that from its presently normal state, with no additional drugs, the pipe or threshold might be raised slightly by the "weak" solution so that a slightly stronger stimulus may be given without an overflow causing pain. In other words, perhaps in this case, soft tissue incisions could be made without pain, but the extraction of a tooth would produce enough stimulus to cause an overflow; therefore, the concentration of the solution must be increased to the "medium" or "strong" in order to raise the pipe or threshold of pain so that the increased stimulus may be tolerated without pain.

Sometimes the infection has so sensitized the sensory nerve fibers that even our "strong" solutions will not raise the threshold high enough to render the patient free of torture. It is in these cases that the operator often loses the confidence and respect of his patient. Here, truly, is a need for general anesthesia.

Again, we are often confronted with the question, "What concentration of epinephrine should I use in my anesthetic solution?" Very often there are differences of opinions and theories in answer to this question. There are those who definitely believe that the stronger concentration of a vasoconstrictor should be used. To uphold their argument, these men state that more profound anesthesia is acquired in addition to a more nearly bloodless field. Those operators

who uphold the theory of milder concentrations of the vasoconstrictor state that the bloodless field is too much so, causing dry sockets and other complications.

There is ample evidence that greater concentrations of vasoconstricting agents will not only give a field less saturated with blood, but also give more

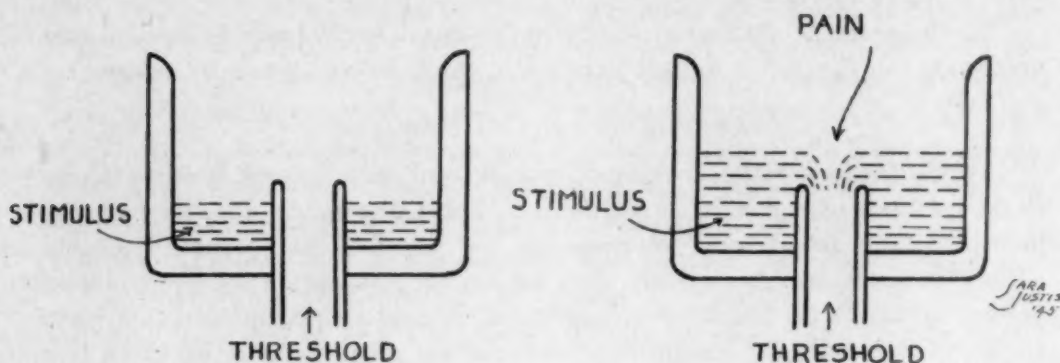


Fig. 15.

Fig. 16.

Fig. 15.—Position of the threshold of pain with normal stimulus.

Fig. 16.—Position of the threshold of pain with increased stimulus. This shows an occurrence of pain.

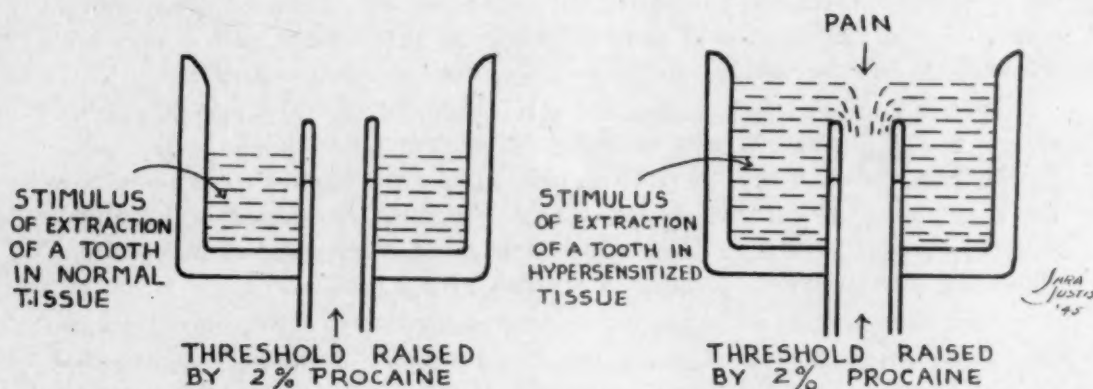


Fig. 17.

Fig. 18.

Fig. 17.—Threshold of pain raised by 2 per cent procaine, thus bringing it above the increased stimulus.

Fig. 18.—The threshold of pain is not raised high enough by 2 per cent procaine to withstand the stimulus given by the extraction of a tooth in a hypersensitive condition.

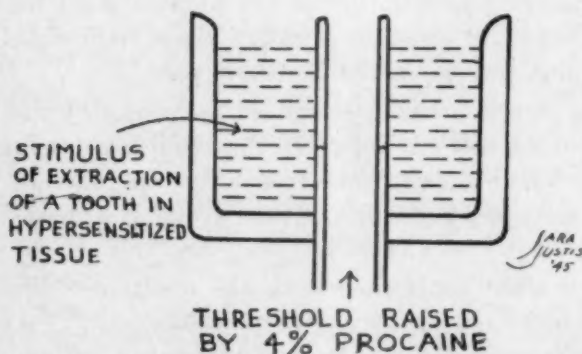


Fig. 19.—Four per cent procaine raises the threshold above the stimulus given by a hypersensitive condition.

profound anesthesia with a duration of greater length. That, of course, is said in praising this type of anesthetic solution; however, in the negative it is true that more often postoperative conditions present themselves in greater severity when the operation is performed under anesthetics containing greater concentrations of a vasoconstrictor. One theory behind this is that with excessive vascular constriction the "normal" amount of toxin given off by the extraction of a tooth is withheld in the socket and may remain one to two hours longer than would otherwise be the case. If a smaller amount of the vasoconstrictor had been used, the body defenses would have long before been in action, thus removing the disturbing agents before concentrated infection occurred. This probably is the reason, also, why greater success and fewer postoperative disturbances are encountered while operating under general anesthesia.

It may be worthy, therefore, at this time to say that there are perhaps good and bad points in both instances. If the patient is one of long standing and the operator knows his general condition by previous experience with him, his postoperative complications usually suffered, if any, and the ease with which anesthesia may be effected, then perhaps a wise choice could be made; this choice varying, of course, from one side to the other, according to the condition of the patient. For example, if the patient has a chronic abscess, or for some other reason there is particular difficulty in acquiring anesthesia, it would be a wise choice to use the stronger solution of a vasoconstrictor. On the other hand, if the patient is one who has been routinely troubled postoperatively, then the wise choice would be that of an anesthetic containing a smaller concentration of the anesthetic and vasoconstrictor so that the chances for postoperative complications may be considerably diminished.

TOPICAL ANESTHESIA

Very often questions are asked by the patient, such as, "Do I have to have the needle?" or, "Can you extract it without the needle?" These questions should be sufficient evidence that the average patient greatly fears the needle. Most of us know by experience that the needle, in most instances, does not actually give unbearable pain, yet the amount of pain given seems to be sufficiently magnified in the mind of the patient to cause exaggerated hypertension and extreme nervousness.

For the above reasons, and perhaps many more, it is a wise move on the part of the operator to utilize medicaments effecting topical anesthesia. These, if properly used, will at least admit the bevel of the needle beneath the surface without pain. At this time the deposition of a drop or two of the anesthetic solution should be made every few seconds while slowly advancing the needle, thus always anesthetizing the tissues slightly in advance until the desired depth is reached. Shortly the patient will realize that he has not been hurt and will become much more calm, greatly facilitating the operation.

These points, as small in importance as they may seem, play an important role in advancing the practice of the young surgeon. Therefore, it is only logical that every operator, both experienced and inexperienced, should strive to acquire a technique of injection that will place the patient at ease from the beginning of the operation. If this is done, anticipation of further pain is readily diminished.

There are several good topical anesthetics on the market available for our use. Most of them effect anesthesia by their application in a fairly concentrated

form on a pellet of cotton to a dried surface. This being held for a short period of time will diminish the sensitivity of the area and a shallow puncture of the tissue may be made with little or no pain.

Very good results may be obtained, when topical anesthesia is desired, by the use of ethyl chloride. This is a highly volatile liquid, due to its low boiling point; therefore, when sprayed on the tissues, an extremely frigid condition is acquired and sensitivity is obliterated. The container should be held at such a distance from the tissue to give a frosty appearance within three or four seconds, at which time the needle may be introduced unknown to the patient. This technique is of exceptional value when performing palatal injections where the tissue is not only particularly sensitive but very dense.

Anesthetics for topical application most popularly used today are 10 per cent butyn, 5 per cent cocaine with 4 per cent phenol, and 5 per cent amylcaine, all of which give appreciable results when applied with slight pressure. There are several others used that have been reported to give better results than the above-mentioned ones. However, almost all of these seem to do so more by chemical cauterization than by actual anesthesia; therefore, these are contraindicated.

Anesthesia given by the topical method would more closely approach the ideal. However, perfection is quite some distance away at the present time.

AIDS IN GENERAL ANESTHESIA

One meets with considerable controversy very often when advocating general anesthesia to be used frequently in the practice of exodontia and oral surgery. A few incidences where general anesthesia has a great advantage over the local form are:

1. To reduce stubborn dislocations. Very often, in hospital work, mandibular joint dislocations are presented which seem to be impossible to reduce by force with the patient in a conscious state. This is due to the patient's anticipation of pressure and his muscular action delivered against this applied force for protection against further pain. Some patients are not capable of complete relaxation under a mental strain. The application of general anesthesia is the answer to this problem, for hardly any more than the excitement stage must be reached before the mandible may be easily and quickly pressed back into the correct position. This procedure saves a great amount of time and reduces post-manipulative soreness.

2. In dealing with frightened children: It is unfair to children, particularly those who are afraid and at the same time too young to realize that treatment must be administered, to force them amid tears and crying to accept the puncture of a needle, and then to watch, what is to them, weird-appearing instruments flash before them. If the extraction to be done is one that will apparently require anesthesia of only a few seconds, administration of ethyl chloride through a piece of gauze about eight layers thick over the nose and mouth will rapidly place the patient under adequate anesthesia for this operation. Induction and recovery alike are rapid and seldom is nausea severely experienced. This form of anesthesia is not recommended for lengthy operations—it should be applied only in extremely short cases.

The use of vinethene has greatly facilitated child exodontia. Though not as rapid as ethyl chloride, vinethene has a fast induction time and is eliminated rapidly from the blood, giving in most cases ideal, uneventful recovery. Vine-

there may be used on cases of longer duration, and it is much safer than ethyl chloride, being considerably easier on the heart. The equipment needed for administration is practically nil, making it ideal also under exceptional conditions. Vinethene is an ether, but is slightly more volatile, accounting for the fact that it renders faster induction and recovery. Its elimination is through the lungs and it is unchanged in the process.

3. In extremely nervous patients: These patients are usually in the class of those who are old enough to fully realize the value of treatment, yet are unable to control their fears and reactions. For these patients nitrous oxide and oxygen are ideal for application. The induction is not disagreeable and the patient falls into deep sleep. If food and drink are omitted for approximately three hours before administration, nausea seldom occurs.

4. In establishment of drainage for cases of osteomyelitis and abscesses: This indication is due greatly to the inability to get profound local anesthesia safely. As was described before, the threshold of pain in these cases is very low and the nerve fibers are extremely sensitive, making complete anesthesia by local means practically impossible. The application, therefore, of general anesthesia, being fair to the patient, is the only rightful method by which these patients may be treated.

5. For extraction of teeth held by inflamed or infected tissue: The reasons here are the same concerning hypersensitivity and lowered threshold of pain. Often local anesthesia is effective in these cases but seldom ever enough to give the patient complete absence of pain.

6. For multiple extractions: It is reasonable to believe that the body defenses can easily take care of tissue reaction locally if they are of a small area; however, if the area is increased, more strain is thrown into the fight and the etiology is much harder to overthrow. For these reasons, multiple extractions are seldom advised. When this particular procedure is undertaken, however, general anesthesia is advised. We are often eager to state that local anesthesia will give a bloodless field; yet, seldom do we realize that a bloodless field is our severe enemy and will occasionally lead to much trouble. Hampering blood supply for the best part of an hour, as is often done with local anesthesia, weakens the tissue defenses in that area, and infection rapidly takes advantage of these ideal opportunities offered; this can give rise to one or more of any number of infections—the most prevalent being dry sockets. General anesthesia does not decrease the flow of blood; therefore, the tissues, at all times, receive a normal supply, feeding them with the necessary foodstuffs and carrying away the toxins given off here. All of this combines to bring about normal tissue reaction and hastens healing. Delayed healing most often brings on pain and infection, both of which are dreadful to the patient and annoying to the operator in charge of the case.

In order to appreciate the points listed above, it should be kept in mind that the operator must be at least moderately capable of administering general anesthesia. It is seldom ever necessary to deviate from the normal technique of administration, but when it is found advisable, these deviations must be well founded and mastered by the anesthetist.

Under my observation, much smoother anesthesia has been obtained by using the technique of first saturating the patient with oxygen. It is particularly good with alcoholics and drug addicts. This method seems to delay anesthesia only slightly, if at all. The simple technique is as follows: The patient

is given 100 per cent oxygen for approximately twelve inhalations. At the end of this time the normal dose of the anesthetic agent should be given. For example: if nitrous oxide is being used, the machine would be regulated to nitrous oxide 80 per cent and oxygen 20 per cent. The nitrous oxide, of course, many times must be increased, and very much so with difficult patients.

The above technique is not recommended for very young children, due to their extreme nervousness and hypertension.

PREANESTHESIA SEDATION

Another important factor that should not be overlooked is preanesthetic sedation. In my opinion this subject is much too important to be discussed so little, as it usually is with the student-surgeon. This is not only an aid in calming the patient, thereby giving him more confidence, but also an aid to the action of anesthesia. Heavy sedation of the dental patient in the office is not advocated here, but a normal or light sedation is of great value. With preanesthetic sedation the metabolic rate is lowered, thus greatly facilitating the procedure.

The average preanesthetic sedation will last from two to four hours, leaving the patient in a state of stupidity for the same length of time, thus making necessary an escort for him if he is allowed to leave the office before recuperation.

I am not at all in accord with the opinion of those operators who state that preanesthetic sedation is of little use or value on children. An advocacy is made here, and I am sure that the majority of skilled anesthetists would agree, that in these cases, above all, correct sedation should be given for safety and ease in handling young patients. The child will undergo a much reduced excitement stage and the anesthetic recuperation will be smoother.

Our preanesthetic agents now of most value may be placed in two classes: First, derivatives of opium, which are the most important of the group. Morphine is active both as a hypnotic and analgesic; it acts very smoothly in reducing the rate of metabolism. The second group is that of the barbiturates. While not as important as a whole as the first group, these are of more importance to the dentists for office operations. They are seconal, nembutal, and luminal. In the dental office and clinic I prefer to use nembutal because of its smooth action and less severe onset. In hospital operations I prefer the use of seconal because it is rapid in onset. Then for postoperative sedation luminal is preferable because of its long-lasting action. The dosage for these drugs is from $1\frac{1}{2}$ to 3 grains.

AIRWAYS

Many difficulties and deaths in anesthesia are caused by obstruction of the air passage, such as the tongue falling back and closing the air passage, blood and blood clots, mucous secretion, malposition of the head, throat packs and extracted teeth which have escaped from the operator and dropped back into the air passage.

A case comes to my mind where the only child of a prominent family was taken to the family dentist for the extraction of a loose deciduous tooth. Gas was advised and the child was placed in the hospital. The anesthetic was given and the extraction was so simple that the dentist proceeded to lift the tooth out without a mouth pack. The tooth slipped from the forceps and dropped down the throat into the trachea. Several methods were used, but removal could not be successfully executed, and the child died on the table.

There are two points in this case that should be noted. The first is that, regardless of apparent simplicity, a throat pack should be used. The second point is rather strange



Fig. 20.—Position of the sandbag under the patient's shoulders in order to keep an open airway.

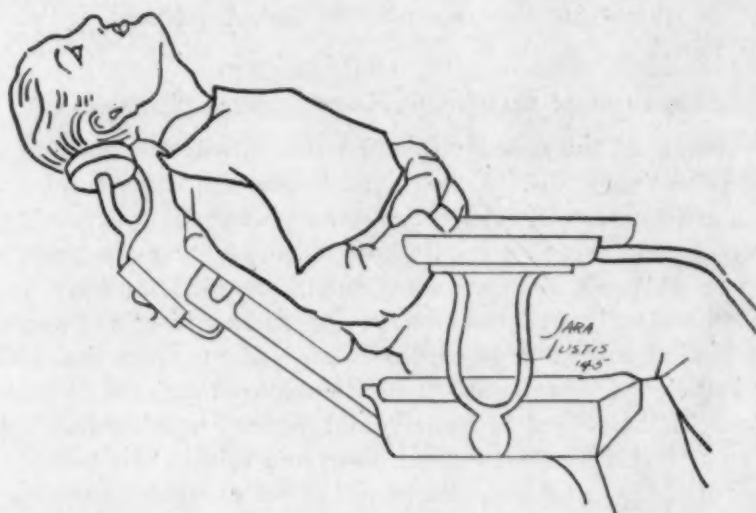


Fig. 21.—Correct chair position of a patient in order to keep an open airway under general anesthesia.

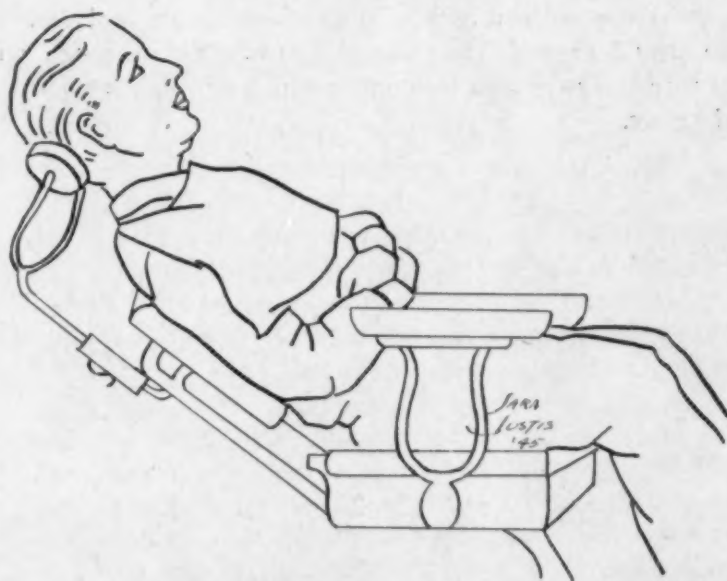


Fig. 22.—Incorrect chair position of a patient under general anesthesia.

in that even though the dentist advised the best of everything for this patient by advocating general anesthesia and hospitalization where all facilities and best of care could be utilized, he, the operator, failed to use that one bit of knowledge that is so often placed before us, "use the throat pack in every case of general anesthesia."

There are several ways of keeping an airway open. The chin should be extended or away from the chest. To do this, a sandbag may be placed under the patient's shoulders, thus allowing his head to drop back, if the patient is in a reclining position on the operating table. While the patient is in the dental chair, the chin may be held up with the fingers of the hand which is holding the mask in place. A suture may be placed through the tongue as a means of holding it out of the throat. Also the tongue forceps may be used for this purpose. In many cases artificial airways are used through the nose to assure good air passage. Intratracheal anesthesia is advised when operating on lengthy cases. This is done by passing a fairly large tube through the nose down the throat and into the trachea, thus assuring an open airway. With this condition prevailing, the throat may be completely packed off and anesthesia given through the tube.

PRECAUTION AGAINST EXCESSIVELY DEEP ANESTHESIA

While operating, the surgeon should watch closely the color of the blood, the lips, and the fingernails. These in particular are easy for observation because such a great change in color occurs during even slight cyanosis that it can be easily recognized. Cyanosis should be avoided, and when it occurs immediate action must be taken. If the cyanosis is due to an overdose of gas, pure oxygen must be given instantly, and the airways opened as widely as possible by positioning the head and neck. However, if cyanosis is due to an obstruction caused by foreign bodies, the throat pack should be removed and the patient subjected to aspiration. If the patient is a small child, he may be immediately picked up, placed over one forearm with the head down and held in this position while the index finger of the anesthetist is placed in the throat so that the tongue may be held clear of the airway.

CONCLUSION

Our modern anesthesia is not ideal, but we are now able to extract teeth and operate otherwise without pain. With these agents and facilities at our command, we should exercise their use and should not, through hurried procedures, eliminate the steps and techniques which will deliver the patient from that dreaded agony.

GINGIVAL SYMPTOM COMPLEXES

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INTRODUCTION

IN MANY cases of gingivitis full clinical and laboratory findings are necessary for a complete and accurate diagnosis, prognosis, and for satisfactory treatment. Inflammations of the gingivae per se are seldom clinical entities. More often they are symptom complexes, the diagnoses of which will include recognition of various systemic diseases. The dentist, therefore, should direct his attention not only to the manifestations that may be caused by local irritants but also to the symptoms that may be diagnostic of disease elsewhere in the body. Local factors, such as trauma from malocclusion, calculus deposits, food impactions, and misuse of the toothbrush, as microorganisms harbored in the subgingival spaces or under third molar gum flaps, as various chemical agents taken into the mouth, or as excessive local thermal changes are not to be minimized in their etiological importance. But due recognition should also be made of such clinical manifestations as fever, rapid pulse, superficial adenopathy, purpura, and others that may be diagnostic of systemic disease. The diagnostic laboratory is often helpful in revealing such symptoms as anemia, leucocytosis, leucopenia, hyperglycemia, glycosuria, a positive Kahn, or heterophile antibody reaction. In some cases a history obtained from the patient will suggest a systemic disease that is complicating the inflammation of the gingival tissues. The severity of both local and systemic findings then should be carefully evaluated in determining the prognosis and treatment of the gingivitis. These may be of such a nature that correction can be readily made, thereby indicating a good prognosis, while, on the other hand, regardless of local treatment the gingivitis may persist because of some very serious systemic disturbance.

For example, if general metabolic or infectious diseases of known etiology, such as diabetes or syphilis, if blood diseases such as infectious mononucleosis or the leucemias, or if various allergic conditions and dermatological diseases, such as pemphigus of uncertain etiology, are present, then the gingival tissues will suffer concurrently with other parts of the body. Therefore, we must not only be concerned with the harm infected diseased gums may bring to regional structures by extension and to remote parts of the body through body fluids, but also with the harm systemic disease may bring the gingivae. We must realize that both considerations are quite important and warrant close observation and study. The earliest signs and symptoms of certain fatal systemic diseases may be manifested in the gingivae or oral mucosa, and the patient will seek first the advice of a dentist; thus, he is afforded an opportunity to aid in the early diagnosis of serious conditions. More early diagnoses would be valuable to practitioners in ascertaining how much earlier treatment might produce a more favorable prognosis. In many instances the physician does not see the patient until the disease is well advanced.

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The objective of this paper, then, is to show how certain clinical and laboratory findings are of value in making a complete and accurate diagnosis and prognosis in gingival symptom complexes so that rational treatment may be determined and administered. The writer will attempt to accomplish this aim by discussing literature citations and by describing and illustrating briefly a few of the many gingival symptom complexes the dentist may encounter in his practice.

DIABETES MELLITUS

There are many diseases of metabolism which may complicate a gingivitis. In some of them the exact disturbance is fairly well understood, while in others it is uncertain. As an example of one that is fairly well understood, I have selected diabetes mellitus.

This disease is characterized by the failure of the tissues of the body to oxidize carbohydrates at a normal rate. This failure is due to a lack of "available" insulin which most probably is an internal secretory product of the pancreas. Further, there is defective glycogen storage and hyperglycemia with resulting glycosuria, and secondary disorders of fat and protein metabolism develop. All grades of severity are found in clinical diabetes, indicating varying degrees of impaired insulin production. The oral manifestations are probably due to the dehydration and the high sugar content of the tissue, which lower the tissue vitality and its resistance to infection. Of the three main groups of complications in diabetes, namely, cardiovascular disease, infections, and acidosis, infections which may originate in the mouth are very important and require prompt treatment.

The clinical findings in the mouth which should be ascertained are xerostomia, sweetish taste, acetone breath, marginal inflammation of the gingivae, and periodontal disease. This involvement of the investing tissues of the teeth may be present in the absence of many of the local irritants the dentist commonly looks for. Gingival calculus, however, is one local agent that is often present in diabetes. Frequently there are heavy tartar deposits, usually of the supragingival variety. The bacterial flora of the gingival exudate is quite variable, but may show what is regarded as Vincent's flora. It is especially important to keep the flora at a minimum because of the danger of spread of pathogenic bacteria throughout the body. Checkup microscopic examination of gingival smears we have found to be helpful in knowing to what extent control is being secured.

In Fig. 1, N. P., a white woman, aged 24 years, a case of inadequately controlled diabetes is seen. The gingivae were swollen, red, and spongy. Supragingival calculus was abundant. The teeth were loose and the investing soft tissues were tender and sore on manipulation. Gingival abscesses formed and polypoid proliferations were growing from under the free gingival margin. The patient was almost blind due to eye complications. Bilateral cataracts of the cortical type sometimes develop in young persons who are subjects of severe diabetes. This patient gave a history of dental care over a period of several years, during which time she was not aware of her diabetic condition. Had her dentist suspected this systemic complication and obtained consultation with a physician, the complete diagnosis could have been secured much earlier, and proper systemic as well as local treatment instituted at a time when only the gingivae were involved. With proper control of the underlying systemic disease, the extensive involvement of the entire periodontium might have been prevented. When we saw the case, however, the morbid anatomy of the hard and investing tissues had progressed to the stage that full-mouth extractions were necessary. Over a period of time, as the patient's condition would permit, the surgery was completed and full dentures made. When the dentist suspects diabetes

in a case giving a negative history of this disease, he should obtain consultation with the physician that urinalysis, blood chemistry, and other procedures might be carried out to establish a complete diagnosis so that proper treatment of both the systemic and oral diseases can be instituted.

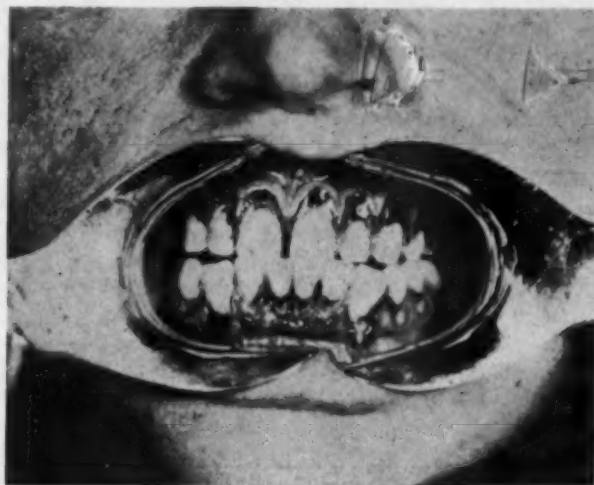


Fig. 1.—N. P. Diabetes mellitus with marked destruction of the investing tissues of the teeth and pathology of the eyes.

SYPHILIS

As an example of an infectious disease of known etiology that lowers the vitality of the gingival tissues and predisposes them to active inflammatory processes that will not ameliorate with local therapeutic treatment alone, we should think of syphilis.

This disease is highly infective in the primary and secondary stages and the lesions of these phases occur in the oral tissues. The primary lesion is followed by the so-called fever of invasion associated with a sore throat and angina. Concomitant with these symptoms the oral mucous patches develop. Sometimes these secondary lesions may be discrete, oval, rounded, grayish-white or reddish-pink patches. In all suspected cases, an attempt should be made to establish the diagnosis. Oil immersion microscopic examination of a dried mixture of a little of the tissue juice from suspected oral chancres or mucous patches, with Gunther's India Ink on a glass slide will reveal the *Treponema*, if present, as fine white spirals on a black background. They are most readily detected, however, in the tissue juice when examined with dark-field illumination.

As *Treponema pallidum* is distributed to all tissues where there are capillaries, the soft and hard investing tissues of the teeth are especially likely to suffer in the tertiary stage of this disease. As Meakins¹ writes, "It may be stated as an axiom that syphilis should be considered the possible cause of every chronic lesion or group of symptoms until proved to the contrary." Two important clinical findings, headache and pains in the bones which are particularly severe at night, should be suspected as being syphilitic. The pathologic processes in congenital syphilis may simulate those of the secondary and tertiary stages of acquired syphilis. We shall illustrate this gingival symptom complex by one of our cases.

R. S., a white woman, 28 years of age, presented to the clinic stating that she had a few small cavities but was more concerned about her gums which bled easily and might be the cause of her being underweight. (Fig. 2.) Superficial examination of the mouth revealed

only a few carious lesions and a rather pale gingiva throughout the mouth. She had a full complement of permanent teeth with the syphilitic developmental defects of the incisors and all four first molars. This cardinal sign of congenital syphilis should always call for the determination of the present status of this disease in the patient, because the treatment of a syphilitic gingivitis offers certain important considerations. If the patient is under treatment with the heavy metals or the iodides, and the dentist attempts local therapeutic measures, he will encounter difficulty. The gingival tissues will weep severely especially in the face of iodide administrations. We have seen cases with rather extensive ulceration of the gingival tissues which yielded an exudate containing a predominance of fusospirochetal organisms diagnosed by the dentist as Vincent's gingivitis, which he treated by the topical application of bismuth salts or by intramuscular injections of this drug. Such cases did not respond but, on the other hand, an acceleration of the inflammatory process occurred with the deposition of bismuth in the gums. If the patient is already tolerating as much bismuth as is possible without manifestations of toxicity, to administer more in treatment of Vincent's gingivitis is contraindicated. In this disease of the gingival tissues, much proteolytic degeneration of body cells is occurring, and when the bismuth circulating in body fluids in ionic form contacts these degeneration products it is tied up as a sulfide and is not free to act as a spirochetal agent. Therefore, under these conditions the administration of bismuth appears to be useless.



Fig. 2.—R. S., white woman, aged 28 years. Congenital syphilis with gingivitis and alveolar resorption. Note the syphilitic incisors and open contacts. The left mandibular premolars and molars were extracted a few days previous to the time the photograph was made.

In our case R. S. (Fig. 2), an evaluation of both oral and systemic findings was made. The history revealed that the patient had recently had a positive syphilitic serum test, although she had intermittently been under syphilitic treatment since 5 years of age. She also had a severe keratitis. Full-mouth roentgenograms showed marked alveolar resorption. The gingival crevices harbored many bacteria with the Vincent's flora predominating in some areas. Her diet record revealed many deficiencies, especially vitamins of the B complex and ascorbic acid. It was clearly demonstrated that systemic disease, together with diet errors, was complicating what appeared on superficial examination to be a simple marginal gingivitis. The case went to full-mouth extraction with full denture restorations. Her diet record was carefully evaluated and she was instructed in regard to a proper dietary regime. She was also advised to continue to place herself under the care of a physician for the control of the syphilitic condition. Syphilitic gingivitis warrants careful study and consideration because it is a problem of great importance from a public health point of view.

INFECTIOUS MONONUCLEOSIS AND THE ACUTE LEUCEMIAS

Infectious mononucleosis, more properly termed acute infectious lymphocytosis according to Kolmer,² and the acute leucemias are blood diseases of uncertain etiology which may occur, perhaps more often than is generally thought, concurrently with the gingivitis commonly recognized by the dentist as acute Vincent's gingivitis, also called ulceromembranous gingivitis or trench mouth. The etiology of ulceromembranous gingivitis is uncertain as has been pointed

out by Hirshfeld³ in 1940, in his report on the history of Vincent's infection. In considering the etiology of infectious mononucleosis, Kolmer² states that various bacteria and protozoa have been incriminated, but none has been proved the cause. *Listerella monocytogenes* has commanded most attention, but the supporting evidence is extremely meager. At the present time the alleged experimental transmission of the disease to monkeys indicates that it may be a virus infection.⁴ Also recent investigations of several workers⁵⁻⁷ incriminate the herpes simplex virus as the primary etiological agent in ulceromembranous gingivitis. These workers are of the opinion that the fusospirochetal organisms, commonly called the Vincent's flora, play no essential role in the production of the gingival inflammation, but may secondarily contribute to the tissue destruction. It would appear then that a virus invasion of the tissues is the most probable primary etiological agent in both these conditions and that the oral picture is secondarily affected by the activity of many bacteria, including a predominance of the Vincent's spirochetes and fusobacteria. This explanation might account for the fact that acute Vincent's gingivitis often assumes epidemic proportions, because it is well known that virus infections are highly infective and are readily transferred from patient to patient by contact with the saliva or the secretion from the oral blisters or ulcers which contain the virus. It is true that we frequently see blisters on the lips, gingivae, or buccal mucosa in acute Vincent's gingivitis, or obtain a history of their occurrence preceding the development of the gingival edema, pain, soreness, bleeding, and pseudomembrane formation. Figs. 3 and 4 illustrate these lip lesions.



Fig. 3.

Fig. 3.—C. L. B., white man, aged 29 years. Herpetic lip lesions preceded the attack of Vincent's gingivitis.



Fig. 4.

Fig. 4.—B. J. S., white girl, aged 6 years. Rampant caries, Vincent's gingivitis present. Note the large ulcer on the upper lip.

C. L. B. (Fig. 3) presented on three different occasions at several monthly intervals with the typical clinical and bacteriologic findings of acute Vincent's gingivitis. He gave a history each time of the herpetic lip lesions preceding the attack of gingivitis. The serum always gave a negative test for syphilis of which he feared he was a victim.

In B. J. S. (Fig. 4), a child aged 6 years, the lip ulcer had existed about a week previous to the visit to our school clinics because of an ulcerating gingivitis. The child was in a poor nutritional status and was also suffering from rampant caries of the teeth.

The clinical symptoms of pyrexia, headache, malaise, and cervical adenopathy are commonly present both in cases of acute Vincent's gingivitis and in cases of infectious mononucleosis. The Paul-Bunnell serologic test and blood counts are important diagnostic procedures in infectious mononucleosis. Believing that there are common etiological factors in these two diseases, we undertook a study of the blood picture and antibody reaction of the serum in a series of acute Vincent's gingivitis, and found that in a majority of cases there occurred a leucocytosis due to an increase in mononuclear cells, a fair degree of secondary anemia, and a positive heterophile antibody reaction (the Paul-Bunnell test), in titers 1:128 or higher. The positive Paul-Bunnell test is regarded as diagnostic of infectious mononucleosis.



Fig. 5.—D. B., white man, aged 21 years, presented with clinical findings of acute Vincent's gingivitis, elevated temperature, increased pulse rate. The gingival flora and blood are shown in Figs. 6 and 7.

Figs. 5, 6, and 7 illustrate the case of D. B., a white man, aged 21 years, one of the series. This patient had a herpetic lesion on the lower lip, of a few days' duration. He gave a history of no previous attacks of gingivitis. The typical Vincent's flora seen in Fig. 6 was present in all gingival sulci. Note the characteristic cocklebur formation of mature fusobacteria in the upper area of the illustration. The monocytic, lymphocytic (according to Kolmer²) leucocytosis is shown in Fig. 7. In a normal blood picture rarely more than one large lymphocyte is seen in a single field and also only one out of many fields will show any monocytic cells. Some hematologists would regard the cells seen in Fig. 7 as monocytes rather than abnormal lymphocytes. In either case they are large cells having a single large nucleus. The total number of white blood cells in this case varied between 14,000 and 18,000 per cubic millimeter of blood throughout the acute stages of the gingival involvement. A positive Paul-Bunnell test was obtained in a 1:256 titer. We isolated in this case a pure culture of fusobacteria, but were unable to maintain viable cultures long enough to establish any definite pathogenicity by animal experimentation. The case suggests, however, the possible virus invasion preceding the development of the Vincent's flora and the lymphocytic leucocytosis, which relationships have previously been pointed out in this paper.

The prognosis in infectious mononucleosis (or lymphocytosis) is good. Often the patient remains ambulatory throughout its course just as in acute Vincent's gingivitis. It is important, however, to establish an early diagnosis that the fatal acute leucemias, which are also often characterized by an acute Vincent's gingivitis, may be ruled out. In the acute leucemias, the heterophile antibody reaction is negative. The type of blast cell causing the leucocytosis, whether lymphocytic, myelocytic, or monocytic, can be established by special staining methods, but the most dependable differential diagnostic procedure is sternal bone marrow studies which usually show not only the evidence of

severe anemia, but a preponderance of the white cell precursor that is responsible for the peripheral leucocytosis. Early in an acute leucemia, the increase in total white cells may not be greater than it is in infectious mononucleosis. But all symptoms and findings in an acute leucemia run a very rapid course, terminating in death usually within a few weeks. Therefore, an early and accurate diagnosis is important in determining the prognosis and treatment. The development of oral lesions early in an acute leucemia is thought by some² to be especially characteristic of the monocytic type. Oral lesions, however, may occur in any and all of the leucemias, acute or chronic types, and hematological investigations are important in establishing a diagnosis.

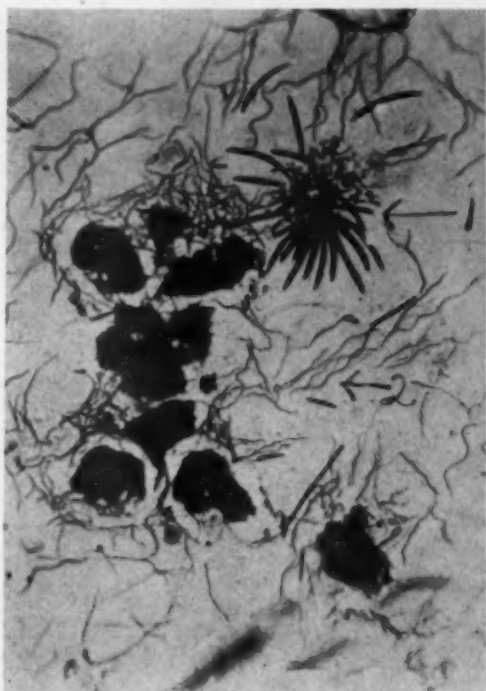


Fig. 6.

Fig. 6.—This photomicrograph shows the Vincent's flora which was present in D. B. in all gingival crevices. 1 is a typical cocklebur formation of mature fusobacteria. 2, The Vincent's spirochetes are very numerous.

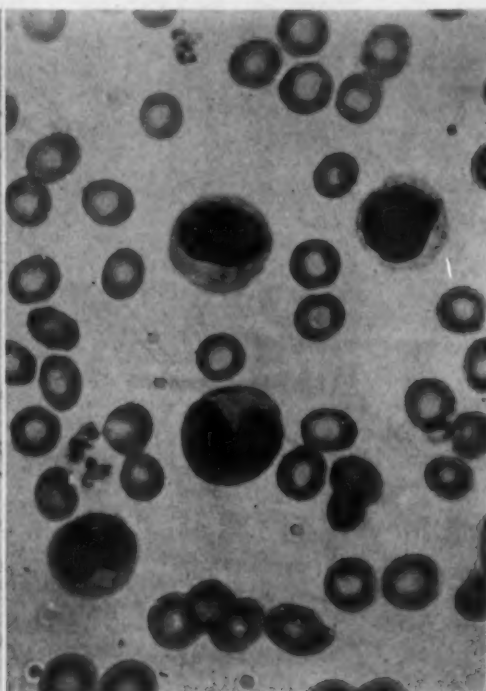


Fig. 7.

Fig. 7.—This is a photomicrograph of a blood smear from D. B. A monocytic (abnormal lymphocytic) leucocytosis was present.

THE PATHOGENICITY OF FUSOSPIROCHETAL ORGANISMS

Since many statements have been made in this paper regarding the fusospirochetal organisms being present in the gingival inflammations complicated by systemic diseases, we wish to pause to comment on the pathogenicity of these organisms.

Some investigators are of the opinion that the fusiform bacilli and spirilla are pathogenic⁸ only when the vitality of the tissue is impaired or when they invade deeper structures or body fluids where the degree of anaerobiosis is high. That these organisms are anaerobic and only facultatively aerobic has long been established by cultural studies.⁸⁻¹⁰ An explanation of their rapid proliferation in the gingival tissues may be that in tissues of lowered vitality, other aerobic organisms, such as streptococci, staphylococci, and diplococci also proliferate rapidly using up oxygen which enhances the growth of the

fusospirochetal organisms which may be found in great numbers in the zone between the necrotic and living tissue.¹¹ In some histological sections, the bacilli alone are seen invading, in others the spirals alone, while in still others both are seen, the spirals being in advance of the fusiforms. Tunnicliff, Fink, and Hammond⁸ have recently shown that immunity tests (phagocytic or opsonic index) on patients with pyorrhea and ulceromembranous gingivitis demonstrate the pathogenicity of fusiform bacilli. That the fusospirochetal organisms may be pathogenic in the blood stream has been shown in our case¹¹ of fatal oral fusospirochetosis complicated by a blood dyscrasia.

Much of our study of gingival smears and cultural work on fusospirochetal organisms points to the belief that there are many species of spirochetes



Fig. 8.—C. C. C., white woman, aged 25 years. Note the ulcerated hemorrhagic gingivae and the fistulous opening on the right cheek.

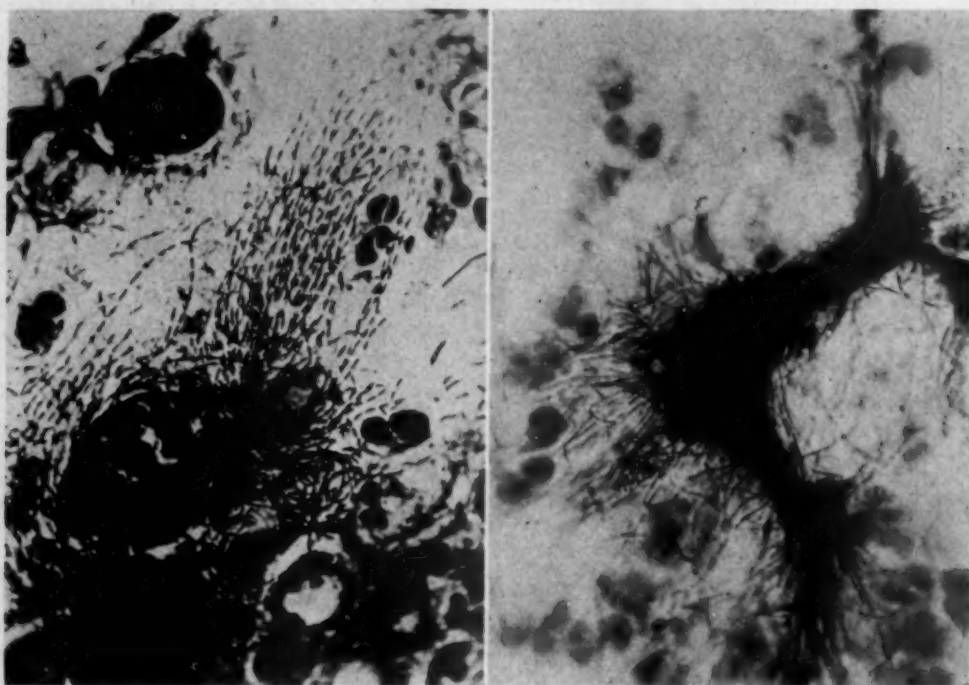


Fig. 9.

Fig. 9.—A photomicrograph of the fusobacteria which predominate in the gingival exudate from the lower anterior and right molar areas.

Fig. 10.—A photomicrograph of the bacteria seen in stained spread of exudate removed from the fistulous tract. Note morphologic similarity to those seen in the gingival exudate, Fig. 9.

and also fusiform bacilli in the mouth, that they are very pleomorphic, especially the fusobacteria which probably have a life cycle¹² comparable to higher bacterial forms, that environmental changes will cause the bacilli to reproduce at different phases of this life cycle so that, under unfavorable conditions, the whole cycle may not occur. The bacilli vary markedly in size as our photomicrographs made at the same magnification will show.¹³ The greater the tissue breakdown and the more unhygienic the mouth, the more the fusospirochetal organisms seem to flourish and sometimes the fusobacteria attain a very large size.



Fig. 11.—Same case as shown in Fig. 8 after treatment. The scar tissue seen at the site of the fistulous opening completely disappeared and little evidence of the previous pathologic process was left.

In our case C. C. C., a white woman, aged 25 years, illustrated in Figs. 8, 9, 10, and 11, the fusobacteria predominating in the gingival exudate was a very small species that occurred in tandem arrangement in parallel rows. An organism having similar morphology and arrangement was isolated from a fistulous tract in the buccal tissues. The patient presented with an ulceromembranous gingivitis and a discharging fistulous tract opening on the cheek opposite the lower right molars. While spirochetes were associated with the fusobacteria in some of the gingival sulci, they did not predominate in any of the areas of the mouth. The fusobacteria shown in Fig. 9 predominated the exudate examined from the lower anterior mouth shown in Fig. 8. The ulcerations were the most severe in this location. This same fusobacteria was also present in the exudate obtained from the molar area opposite the fistulous tract. Clinically, this organism appeared to be the principal pathogen in the inflammatory process involving the investing tissues as well as the soft tissues of the cheek. This patient improved after local gingival treatment, extraction of the teeth with periapical infectious processes, and correction of diet errors of which the greatest was a total absence of vitamin C intake.

PURPURA

The manifestations of purpura, spontaneous hemorrhages or extravasations of blood into the tissues, are often seen in gingival inflammations. Usually its signs and symptoms appear also on other mucous membranes and in the skin. The purpuras are generally classified as hemorrhagic diseases which may be either primary or secondary. More often, they are symptoms of a disease or an abnormal state and therefore secondary. In so far as this condition complicates gingivitis, I shall limit my discussion to the secondary type.

Disregarding mechanical purpura, the important changes responsible for the manifestations of purpura whose causes should be sought are² deficiencies in blood coagulation, and increased fragility and permeability of the capillaries and

a combination of these factors. The deficiencies in blood coagulation may be a thrombocytopenia, a hypothrombinemia, a decrease in fibrinogen, or to the excessive amounts of heparin or other anticoagulants. The increased fragility or permeability of the capillaries is due to insufficient amount of cementing substance between the endothelial cells lining capillary walls. Which of these changes are present may be established by laboratory examinations, such as blood platelet counts, prothrombin time, bleeding time, or coagulation time, the capillary fragility test, and blood and urine determinations of ascorbic acid.

The decrease in thrombocytes in the peripheral circulation is caused by some agent, commonly infectious, or drug that either interferes with the production and maturation of the platelets or brings about an abnormal destruction of them. A decreased amount of prothrombin is associated with functional incapacity of the liver to utilize vitamin K or with a diet deficiency of vitamin K or its failure to be absorbed after ingestion. The increased fragility and permeability of capillaries is due to many agents, notably to a deficiency of vitamin C, to allergy from foods, drugs, and acute and subacute infections.

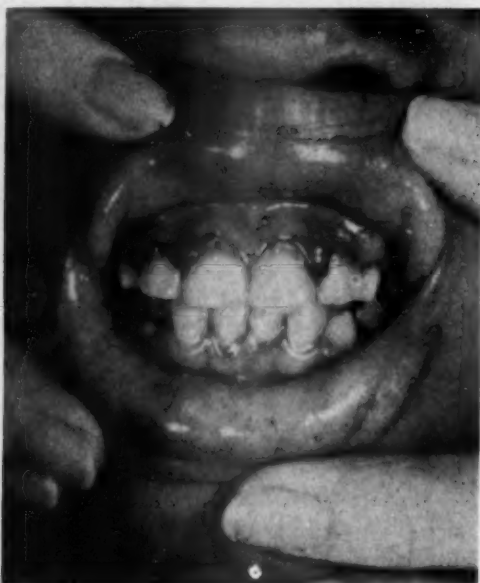


Fig. 12.—A. C., Negro boy, aged 10 years. Purpura complicating gingivitis. Note marginal hemorrhages—one rather large in the right central-lateral interproximal space. Petechiae are present on the nose and lower lip mucosa.

It is important to recognize a purpura complicating an inflammation of the gingivae and base the treatment on the elimination of its cause. With the popular use of the sulfonamide drugs by both the dental and medical professions in the treatment of infectious conditions, caution should be exercised, as these drugs¹⁴ have been shown to develop thrombopenic purpura. Hematological studies should be made during the administration of these drugs to determine any untoward effects on the blood.

In one of our hospital cases, A.C., a Negro boy aged 10 years, shown in Fig. 12,* in addition to the purpuric manifestations on the gingivae, buccal, and lip mucosa, hemorrhages were present in the mucosa of the ears, nose, and throat, and the conjunctivae and the skin showed numerous petechiae. The laboratory examinations revealed a prolonged bleeding time, a low platelet count which became progressively lower during the period of hospitalization, normal coagulation time but imperfect clot formation, all of which are

*This case was studied and reported on by Dr. Homer Vernon Reed, Attendant to the John Gaston Hospital, Memphis, Tenn.

the hematological findings diagnostic of purpura. The capillary fragility test was negative and according to the history the patient had included food containing adequate vitamin C in his diet. We believe that secondary purpura was the condition present and probably due, most immediately, to infection, as the patient was recovering from a very bad cold from which he had suffered the previous week or ten days. No doubt malnutrition also was a factor in the etiology.

LICHEN PLANUS

Lichen planus is an inflammatory dermatosis having oral manifestations. Commonly, the lesions involve the skin of the entire body as well, but Fox¹⁵ reported an interesting series of ten cases in which the eruption was confined to the mouth. The lesions, glistening, reddish or violaceous pinhead-sized papules, at first discrete and later coalescing to form rather sharply defined, rough whitish patches or streaks, may appear on the lips, the gingivae, buccal, and throat mucosa. Other mucous membranes, such as the conjunctival or gastric mucosa may also be attacked. The disease is generally a persistent one and tends to recur even after apparent cure. Sutton¹⁶ has described many aberrant types of eruption which have been given the descriptive terms of annular lichen planus, follicular lichen planus, bullous lichen planus, and others. These different clinical aspects of the disease cause confusion in recognition unless one is familiar with the variations in the appearance of the lesions.

The exact etiological agents of this disease are not known. Like other conditions described in this paper, various bacteria and fungi have been suggested as the cause, but no one has yet identified a specific pathogen. Nervous exhaustion is thought to be an important contributory factor. Dietary deficiencies, especially vitamin insufficiency, have been offered as etiological factors. The disease is more frequent during active adulthood than in any other period of life.

It is important for the dentist to recognize the disease because of the role dental disorders seem to play in its control. We have seen cases in which the lesions appeared first on the oral mucosa and then involved the skin of the entire body, being accompanied by rather severe constitutional symptoms which were unresponsive to all therapeutic efforts until infectious processes involving the investing tissues of the teeth were controlled. Similar findings have been reported by others.^{16, 17}

The prognosis in lichen planus is good, if the treatment is well directed, although it is an exceeding chronic disease. Various drugs have been used to control the eruption, but the correction of all oral foci of infection, and general hygienic measures, such as a well-balanced diet, outdoor life, and freedom from mental worry and care are very beneficial.

PEMPHIGUS

In closing this paper, we have selected pemphigus as our last example of a gingival symptom complex. Particularly the buccal mucosa, gingivae, and nasal mucosa are involved in this disease. Sutton¹⁸ defines pemphigus as an acute or chronic disease characterized by eruption of successive crops of bullae, which develop suddenly, often on apparently normal skin or mucous membrane, accompanied by constitutional disturbances of various degrees, usually resulting in death.

Clinically, pemphigus may be separated into three distinct types: pemphigus vulgaris, pemphigus foliaceus, and pemphigus vegetans. The bullae in the three types differ in clinical and histological appearance, but all are said to contain sterile fluid before rupturing.

In the vulgaris, the bullae are rounded or oval, 1 to 10 cm. in diameter, in number several to a hundred or more, thin-walled, translucent, and sometimes coalescing. They may be umbilicated or irislike. They develop suddenly on apparently normal or slightly reddened areas, increase in size little if at all, and are never infiltrated. In severe cases the lesions may contain blood. The pathology appears to be located between the basal cell layer of the epidermis and the underlying corium, that is, at the dermalepidermal junction, because all layers of the epidermis can be stripped off leaving a denuded painful red corium. The skin covered with firm keratin can be separated only where there are bullae.

In pemphigus foliaceus, large fragile flaccid bullae develop rapidly and soon rupture and form a scabby appearance. There is a diminution of the adhesion between the stratum corneum and the subjacent prickle cell layer and rubbing will not only remove these outer layers of the epidermis over the lesion, but also the corneum of the uninvolved skin between lesions. This clinical finding is known as Nikolsky's sign and is regarded diagnostic of the foliaceus form of pemphigus.

The vegetans bullae, which seldom exhibit any tendency toward healing, involve more frequently mucous membranes, generally of the mouth and pharynx. In this type, the bullae are followed by fungoid growths.

Lever,¹⁸ in 1942, reported on a series of 114 cases of pemphigus in which 62 showed involvement of the oral mucosa, the vegetans form occurring with the greatest frequency, and the foliaceus with the lowest. This finding is probably due to environmental differences. The presence of many bacteria and fungi in the mouth favor the development of the vegetans form. Lever further stated that the oral lesions were the first symptoms of pemphigus in 25 per cent of the cases.

It is important to recognize this disease because its treatment offers many difficulties. Many procedures have been tried and none with any great success because the etiology of the disease is not understood. In summarizing the literature¹⁹ on the causes, it has been stated that the bullae form due to acute lymphatic edema which is brought on by the action of epinephrine, that adrenal insufficiency is a factor, that bacteria are responsible, and that a filterable virus is the agent. Many investigators, however, have expressed the belief that bacteria, when found, represent only a terminal stage. More recent work points to a disturbed metabolism producing the abnormalities of the skin and mucous membranes. For example, MacCardle, Baumberger, and Herold¹⁹ have made histochemical investigations which show that the skin of pemphigus patients contain increased amount of silicon, iron, Manganese, and selenium, and that the chemical consistency of the basal cells of the epidermis is altered, with a resulting elongation of their intercellular fibrils. Their work seems to indicate that the bullae are formed subepidermally in the peripheral third of the dermis where a vesicle forms by coalescence of several vacuoles originating in the dermis among the meshes of the fibrils of the basal cells.

In Fig. 13, J.M.M., an elderly white woman,²⁰ the characteristic lesions of pemphigus vulgaris, as have been described, can be seen on the labial gingivae in the upper mouth. The darker areas were fiery red due to the sloughing off of the epidermal covering which exposed the underlying corium. The patient sought her dentist for relief of the condition involving her gums, at which time she gave a history of the formation of blisters containing water, not only in her mouth, but on the chest, neck, and in the edges of the hair on her scalp. She had suffered from food allergy over thirty-five years, and previous to the appearance of bullae had been on a restricted diet which undoubtedly did not furnish all the essential nutrients. Her dentist advised a high-vitamin diet and therapy,

to which she responded to a limited degree. He also conferred with physicians about the case and every possible medical aid was given. According to hospital laboratory examinations hypothyroidism and probably adrenal dysfunction were present, which endocrine disturbances can be attributed to faulty nutrition. It is known that the production of hormones are dependent upon certain vitamins and proteins and that mineral metabolism is controlled by hormone secretions, particularly those furnished by the parathyroid and adrenal glands. After a duration of twenty-one months of intermittent improvement and exacerbations of the complex symptoms in this case, the patient died. The clinical findings seem to support the belief that pemphigus is due to a metabolic derangement.



FIG. 13.—J. M. M., elderly white woman. Note the darker areas of the labial gingivae in the upper mouth. Here the tissue was fiery red and the epidermal covering was readily removed.

SUMMARY

1. Some of the most common systemic and oral clinical signs and symptoms and laboratory findings in diabetes, syphilis, infectious mononucleosis, acute leucemia, purpura, lichens planus, and pemphigus have been described, and the relationships of these diseases to inflammations of the gingival tissues have been pointed out.
2. The pathogenicity of fusospirochetal organisms, which frequently predominate in the exudate obtained in the gingivitis occurring concurrently in these diseases, has been discussed.
3. The etiological factors of these pathologic processes have been summarized.
4. Attention has been called to the importance of obtaining complete clinical and laboratory data so that both systemic and oral relationships may be understood and rational treatment planned.
5. The various features of the discussion are illustrated by clinical photographs and photomicrographs.

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EXTERNAL SKELETAL FIXATION IN THE DENTAL OFFICE

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IN THE past, one of our greatest problems in fracture fixation has been that of good oral hygiene, due to the fact that intermaxillary wiring has been the chief method employed. It is impossible to give the mouth proper oral care under these conditions, and thus food and foreign material accumulate in the mouth, producing a very offensive odor and unpleasant taste. There are often breaks in the oral mucosa due to trauma received during the accident. Sometimes there are compound fractures, broken teeth, etc., which produce breaks in the oral mucosa. With the communication of the fracture line with the cavity and the accumulation of bacteria, one can readily see why the patient suffers so much pain due to the infection which is present. This type of infection is very difficult to treat because of the handicap of the teeth being ligated.

Another problem of mandibular fractures in which the mandible is immobilized by ligation is that of malnutrition. It has been the experience of the writer that it is impossible for the patient to carry on a normal active routine because of the lack of proper nutrition. While the patient is hospitalized this problem is not so difficult, as the total amount of energy needed is at a minimum. The hospital dietitian has the problem of working out liquid formulas for the patient so that he may receive adequate food for the body.

When the patient is dismissed from the hospital, the problem of malnutrition becomes an important factor. It has been suggested by some writers that in many instances, when the jaws are immobilized, an anterior tooth be removed, thus causing the patient to lose a good tooth; often this was done to solve the feeding problem alone.

There is also the problem of speech when the teeth are ligated. In many cases this must be given considerable attention. In pronunciation the tongue needs sufficient space to form words correctly. As it must be placed at times

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on the incisal edge of the anterior teeth and at other times in the roof of the mouth, one can readily see the importance of the patients enjoying free movement of the jaws.

While reducing many fractures of the mandible, utilizing numerous methods, I have been convinced of the definite superiority of the external fixation procedure. This method meets more of the requirements set up by our profession than any other. Problems such as oral hygiene, dangers of aspiration on regurgitation, nonfunction of the muscles of mastication, normal speech, and normal activity of the patient are reduced to a minimum. As Richison and Kennedy¹ state, "Any procedure, method, or appliance that will reduce the time away from any position, whether defense or military, is a direct contribution to the effort now being made. The use of the skeletal fixation splint in fractures of the mandible has been and is, indeed, a direct contribution to the reduction of time lost by the patient as a casualty. The appliance permits normal masticatory functions and also eliminates any chance of mishaps arising from the closure of the teeth by wire or other appliances placed within the mouth. It allows the patient to carry on his work without pain or discomfort, while the fracture is healing."

Extraoral plaster splint fixation is ideal to use when the patient is edentulous. Richison and Kennedy reported a case in which a skeletal fixation splint was used; also, full dentures were constructed and inserted seventeen days after the skeletal splint was applied, and only nineteen days after the fracture occurred. Occasionally, under my observation, dentures have been reinserted immediately following mandibular fracture fixation, with very good results.

The extraoral plaster splint has been used for some time. It has been suggested and reported by Julious Bourgoyne,² of the University of Tennessee, Oral Surgery Department; also by Julius G. Godwin,³ of the Dental Department, Jefferson Davis Hospital. This simple appliance was first used by Dr. Bourgoyne in the Charity Hospital, New Orleans. The hospital was treating about 35 to 40 mandibular fractures per month, and about 75 per cent of these appeared in the third molar area, in the angle of the mandible, or in edentulous mouths, making it impossible to treat most of these by simple intermaxillary wiring or the use of the open reduction method, as many of them were compounded into the mouth. Therefore, the extraoral method of fixation was in great demand. An application was made in the hospital for several well-known extraoral appliances, and it was found that the expense was too great to meet the demand of the number of fracture cases to be treated. Dr. Bourgoyne then worked out a simple appliance, at first using only one screw on each side of the fracture, and then a heavy wire wrapped around the screws. This method was found to give an adequate fixation in a majority of cases. Then the plaster splint was designed. This method of fixation is in use regularly in the Charity Hospital of New Orleans, in the city hospital of Memphis, the University of Tennessee College of Dentistry, and by a number of dental surgeons.

I have found that this simple method of fixation of mandibular fractures can be used very successfully in the modern dental office. Such cases are reduced in my office devoid of complications. I fully recommend its use in the office of the dental surgeon. The materials that are necessary can be purchased at any surgical supply house and the cost is practically negligible. The materials consist of vitallium screws of the venable type $1\frac{1}{2}$, $1\frac{3}{4}$, and 2 inches long; also, some ordinary wood drill points which can be purchased at any

hardware store, sizes $\frac{3}{32}$ and $\frac{7}{64}$. This diameter is slightly smaller than the threaded shank of the screws. The plaster splint consists of surgical gauze 1 inch wide and ordinary dental plaster or rapid dental stone, and a small roll of 24 gauge stainless steel wire.

To perform this operation successfully in the dental office, there are several preoperative procedures which must be carried out with utmost care. When one is operating in a hospital, the problems of asepsis are few, but if this procedure is done in the dental office there must be some thought and consideration given to sterile technique. With careful arrangement this may be easily accomplished in the modern dental office.

It was found in the beginning with the use of the external plaster splint that the success of the operation depended greatly on the aseptic conditions utilized throughout the procedure. Before the patient is admitted to the dental operating room, the floors should be thoroughly cleaned with some type of satisfactory disinfectant. A sterile towel should drape the headrest of the chair, also the bracket table, and cover any other table or cabinet where instruments might be placed. A complete list should be made of the instruments to be used in the operation, and these should be sterilized, then wrapped in a sterile towel and placed on the bracket table ready for use. In the modern dental office there is no problem in "scrubbing up" and entering the sterile gloves. Anteroposterior and lateral roentgenograms should be studied very carefully, noting the position of the fracture line, the amount of displacement of the fragments, and whether there are any broken teeth or teeth involved in any other way.



Fig. 1.



Fig. 2.

CASE REPORT

The following is a typical case as done in my office: The patient, T. C., Negro male war worker, aged 37 years, suffered an accident in a war plant where he was struck with a wrench while working, producing a mandibular fracture. The patient was taken to the hospital where radiographs were made, and then referred to me. Fig. 12 shows that the mandible was fractured on the right side, beginning at the second premolar and running obliquely distally through the apex of the second premolar. The radiograph revealed on the left side that the fracture began at the third molar and ran obliquely proximally, thus involving the angle of the jaw. I examined the patient twenty-four hours after the accident

and there was some swelling and trismus present; also, some crepitation was noted on digital manipulation. (Fig. 1.) The chief complaint the patient made was that he had severe pain on the right side of the jaw and that he was unable to move his lower jaw as before. I was contacted by the patient's employer and asked to get the patient back to his job as soon as possible. The radiographs were studied closely and the external splint was agreed upon as the best method of fixation.



Fig. 3.



Fig. 4.



Fig. 5.



Fig. 6.

As a preoperative sedative he was given 1 grain of codeine and $1\frac{1}{2}$ grains of secenal thirty minutes before the operation began. After the patient was seated in the dental chair, his face was prepared with alcohol followed with tincture of metaphen. His head and neck were draped with sterile towels which were clamped together. The preoperative radiographs showed that the lower right second premolar and the lower left third molar were in the line of fracture and, therefore, should be extracted. These teeth were removed with no complications. The mandible was palpated and the location of the fracture lines determined. The site for the placement of the screws was ascertained, and the area was infiltrated with 2

per cent novocain solution. (Fig. 2.) Then a small incision, through the skin only, was made with a sharp scalpel at the desired points, as shown in Fig. 3. Blunt dissection with a pair of blunt Mayo scissors was carried out until bone was encountered (Fig. 4). Using a $\frac{3}{32}$ drill point in the straight dental hand piece, a hole was drilled on each side of the fracture line so that the screws would converge at approximately 40 degrees. At first the drill point met with great resistance, due to the outer cortical layer of bone, which somewhat diminished when the marrow cavity was reached. When the drill point reached the inner cortical layer, the resistance became greater again. One word of precaution here is that the drill point must not pierce the mucous membrane of the mouth. Fig. 5 shows very clearly the drill and hand piece being used.

After the drill was withdrawn the screws were inserted; this was done by the use of a screw driver, as shown in Fig. 6. A $1\frac{3}{4}$ inch vitallium screw was placed on each side of the fracture line on the right side. The screws were tightened securely, and then a sterile piece of gauze was placed around the screws next to the skin. This aids in protecting the wound against contamination while the plaster splint is being applied. The screws that were placed on the right side of the face are shown in Fig. 7. I might add that at this point the patient was in a mild sleep.



Fig. 7.

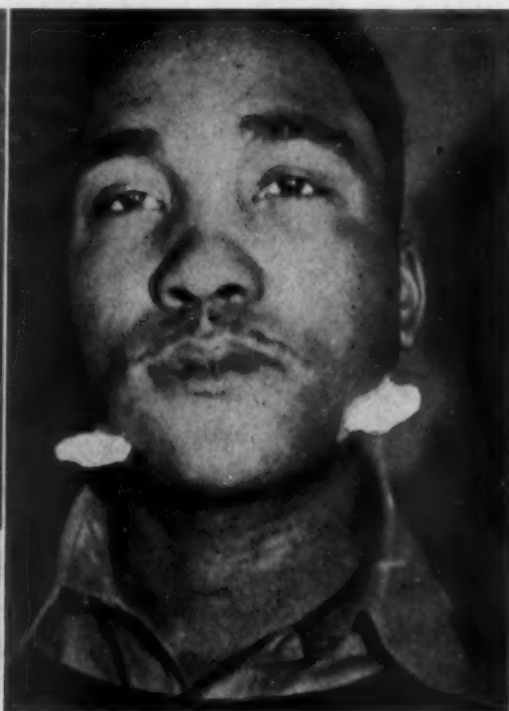


Fig. 8.

The fragments were brought into alignment by digital manipulation and held while a small stainless steel 24 gauge wire was wrapped around the head of each screw; this helped to make the appliance more stable. While the fragments were held in place, a piece of $1\frac{1}{2}$ inch gauze, which had been dipped into a sloppy mix of dental plaster, was wrapped around the screws and molded with the fingers. The fragments were held firmly and in place until the plaster hardened. Fig. 8 shows the plaster splint in place and the operation completed. The total time for the operation was fifty minutes.

The patient was given permission to return to work immediately, and he did so the following day. He was placed on a soft diet for the first two weeks and then given a regular diet as he desired. The patient was seen one week later, when the occlusion was satisfactory. He was instructed to return in four weeks, at which time the appliance would be removed.

The removal of the appliance in the dental office is simple and requires only fifteen to twenty minutes, using no anesthesia. A separating disk was used in cutting through the plaster splint as seen in Fig. 9. The halves of the appliance were unscrewed, causing very little pain. Fig. 10 shows the appliance cut in half just before the screws were removed.

Fig. 11 shows the patient immediately after the appliance had been removed. Note the small area at the site of each screw removal. These healed uneventfully. The patient reported to me that he was very comfortable while wearing the appliance and there was no pain after its application. He had worked every day in his normal routine.



Fig. 9.



Fig. 10.



Fig. 11.

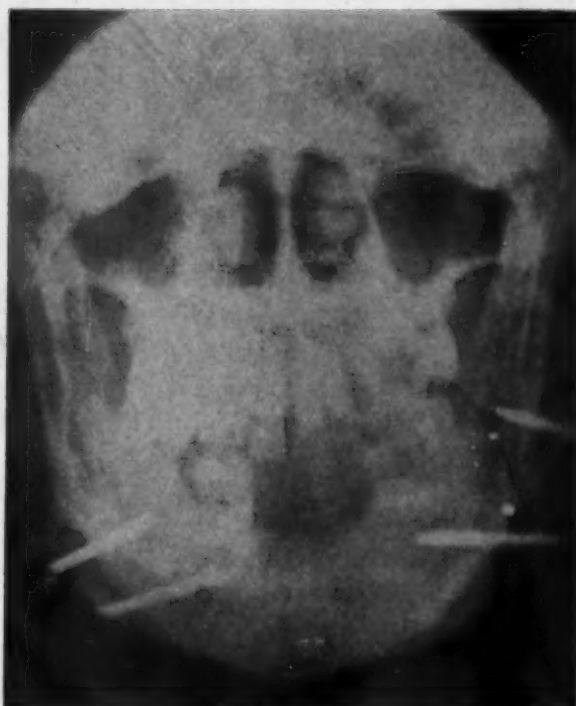


Fig. 12.

The writer has not had develop a single case of osteomyelitis or any infection around the screws. The important thing to keep in mind while doing this operation in the office is cleanliness. As Godwin states, "It may be assumed that an infection would be due rather to the presence of bacteria carried into the bone by the screws, than to a 'foreign body reaction.' " In all cases up to this date the screws, while being removed after the fixation was completed, had to be unscrewed with force; had infection been present, this would not be the case.

CONCLUSION

The extraoral appliance is of great value in correcting many of our previous faults and shortcomings in fracture fixation; therefore, when the need presents itself, this type of fixation should be applied. In the opinion of this writer, its small scar area, more stable fixation, absence of a foreign body contacting the fracture line, better oral hygiene, elimination of dangers of aspiration on regurgitation, normal speech, and normal activity of the patient render it superior to other methods of fixation.

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FIXATION OF PATHOLOGIC FRACTURES OF THE MANDIBLE

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TO THE author's knowledge there has been very little literature concerning the treatment and fixation of fractures resulting from pathologic causes. For this reason, and in answer to a number of requests, this paper has been prepared. The material herein is based upon the observation and treatment of a number of such cases.

The management of pathologic fractures is more difficult than that of traumatic fractures, and the time involved in the procedures applicable to them is considerably longer. In the majority of cases, pathologic fractures result from bone infections such as osteomyelitis. Extractions of teeth or severe abscesses usually lay the foundation for the development of the osteomyelitic type. A good number is also represented by those which are produced by the damaging and debilitating effects of bone cysts and tumors which may also be infected.

Before the fracture is treated, sequestra or foreign bodies, if present, must be removed and infection cleared up completely. (Figs. 1 and 2.) No type of fixation or repair should be attempted until the area is absolutely clear of pus, and until all evidence of drainage has disappeared and has been absent for some length of time. Only after these measures have been taken should the proper treatment be instituted, and consequent successful end results be expected.

The methods of fixation that may be used for fractures of this type with best results are as follows: (1) Open reduction with wires. (2) Intermaxillary

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wiring. (3) Skeletal fixation. A bone graft may be used with any one of these methods. The open reduction method, using a plate and screws, is omitted from the above-mentioned methods because the strip of remaining bone along the lower border of the mandible, after this type of fracture occurs, is usually rather thin and will not tolerate the massive amount of metal.

The method of fixation to be used for pathologic fractures will depend largely upon the amount of bone structure which has been lost. If the loss has been slight, and if the fragments of the lower border of the mandible remain in close proximity, as is the case many times, no bone graft is needed and the bone ends may be placed in direct contact and fixed. If the bone loss has been extensive, a graft must be used.

Fig. 1.

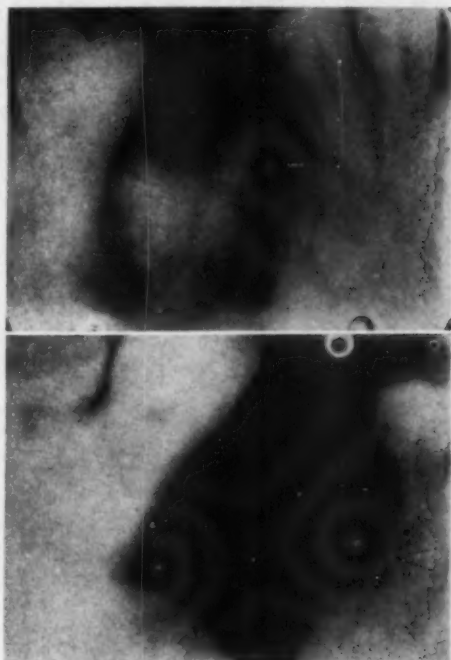


Fig. 2.

Fig. 1.—Radiograph of the first molar area of the mandible showing a large sequestrum in the line of a pathologic fracture, which resulted from an abscess of the tooth previous to its removal.

Fig. 2.—The same area as shown in Fig. 1 after removal of the sequestrum intraorally. Note the increased firmness of the bone surrounding the second premolar.

The cystic type of fracture usually breaks through the upper border, or alveolar process, leaving a lower border of thin bone. The break then generally occurs as a result of some light, unintentional blow to the area, or while the patient is masticating his food. This type of fracture may be repaired, using the extraoral method, after the cyst has been removed without waiting for the mucous membrane to heal, providing the pins or screws are placed outside the area of the incision so that they will not communicate with the mouth. The mucous membrane must be allowed to heal, after the removal of the cyst, if the open reduction with wire is used.

Fractures occurring in bones that have been sites of malignant tumor growths, that have been operated upon and removed, usually require bone grafts. This is due to the fact that often a large amount of bone has been removed as a safeguard against the recurrence of the malignant growth. A

bone or soft-tissue graft should not be done for approximately two years after the removal of a malignant growth in order to allow the area all the benefits of its recuperative powers.

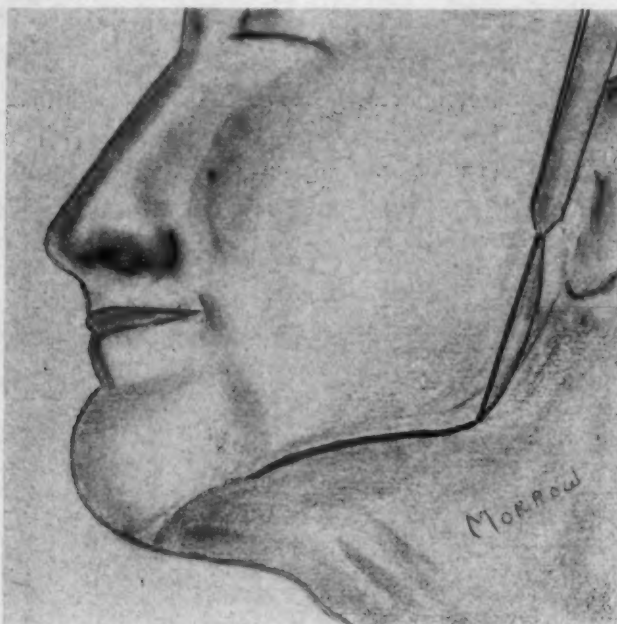


Fig. 3.—Incision shown along the lower border of the mandible in the shadow of the jaw. The incision placed in this position will leave little visibility of a scar when healed.

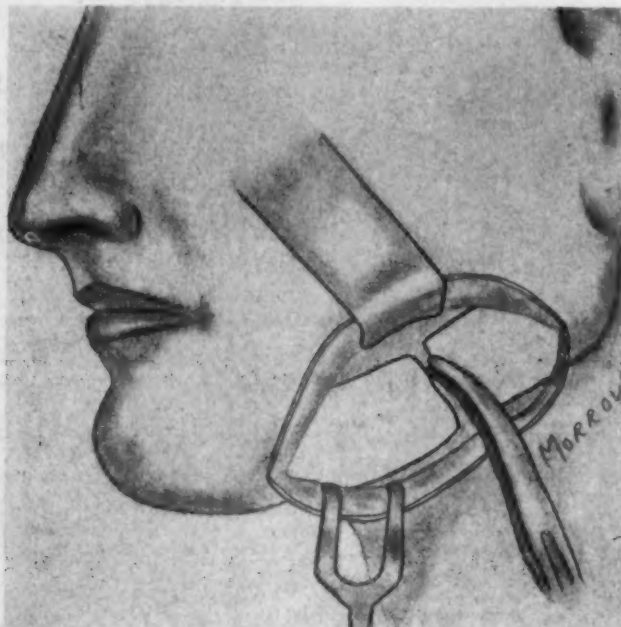


Fig. 4.—Application of the rongeurs to freshen the bone ends.

TECHNIQUE FOR OPEN REDUCTION WITH WIRES

An incision is made through the skin, along the lower border of the mandible in the shadow of the jaw (Fig. 3). With the aid of the Mayo scissors and the periosteotome, the deep tissues are retracted from the bone. If the area of operation is in the region of the external maxillary artery, it may be located and

tied off with No. 50 cotton thread or 00 plain catgut before it is severed. At no time should the mucous membrane of the mouth be perforated. The two approximating extremities of the bone are freshened so that the raw surfaces may be in contact with each other. (Fig. 4.) One-fourth inch on each side of the line of fracture a small hole is drilled with a hand drill containing a 3/32 drill point. A piece of 28 gauge stainless steel is passed through the holes and the bone ends pulled together. (Fig. 5.) The operator should be absolutely certain that the bone ends are in perfect contact and that no tissue lies between them. Bone growth can span a small gap, but any intervening tissue would be detrimental to regeneration. The wire is twisted and the ends cut to the length of 1/4 inch and laid flat against the bone surface. The operator is reminded not to enter the mouth in any way through the extraoral incision. The wire is a foreign body across the line of fracture. If this becomes contaminated it will provide a continuous source of bacteria, and if there is a subsequent development of infection in the underlying bone and overlying tissues, no bone regeneration would be likely.

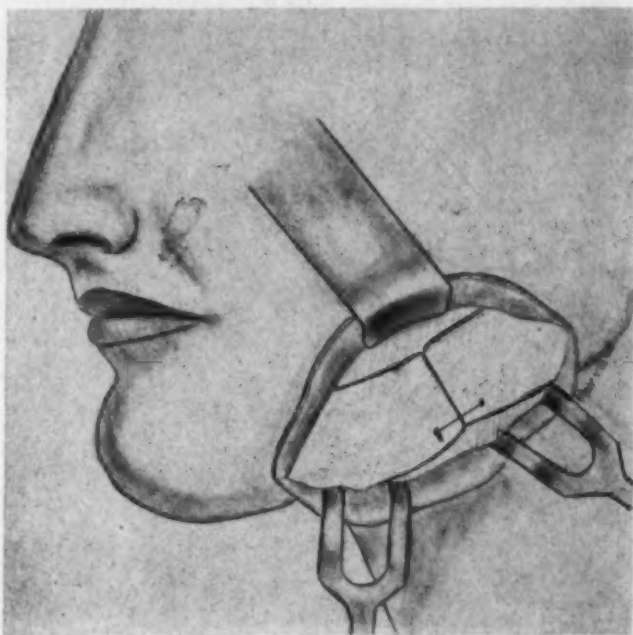


Fig. 5.—Position of the wire to hold the bone fragments in contact.

The deep tissues are approximated in a normal position with 00 plain catgut or No. 50 cotton thread, using interrupted sutures. Superficial sutures are taken with the same material. The skin should be so well approximated so that there will be no tension on the sutures. Medium black silk or horsehair sutures are used for the skin. A small gauze sponge is then placed over the incision, and this is covered by a larger sponge which is taped to the face. The large sponge should be removed and replaced daily for three days without disturbing the small one. On the fourth day, the alternating sutures are removed. The dressing is changed daily until all dried blood clots have disappeared from the skin, at which time dressings are no longer necessary.

During the entire postoperative course, this period of time being at least five weeks, the patient must be kept on a strict soft diet so that the part may be kept immobilized.

TECHNIQUE FOR INTERMAXILLARY WIRING

The treatment and preparation preceding intermaxillary wiring can usually be given by using the bone file to freshen the bone ends intraorally, but this intraoral technique does not always yield the best results. Wiring cannot be used successfully if there are not enough firm teeth on both the anterior and posterior sides of the fracture site.

If extraoral treatment is to be given, the procedure of freshening the bone ends is the same as that used in the open reduction with wires. After the area is closed and sutured, the teeth of the lower arch should be wired to the teeth of the upper arch in order to immobilize firmly the bone fragments involved in the fracture (Fig. 6).

Results reported from the use of intermaxillary wiring are unfavorable, and it is not generally accepted as a method of choice for such cases.

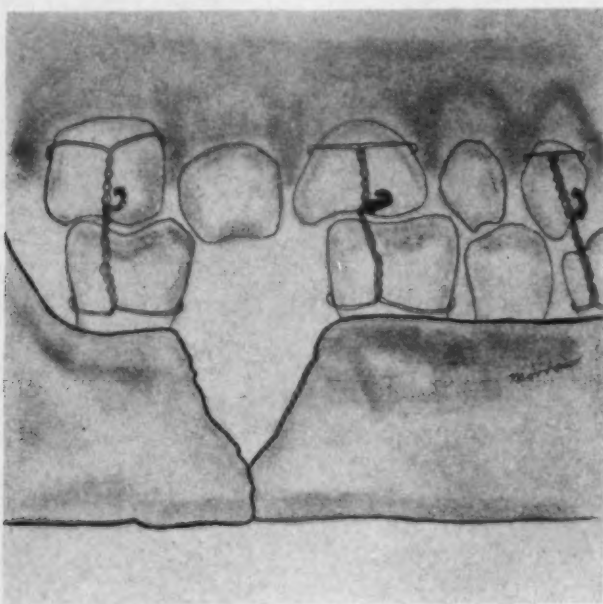


Fig. 6.—The position of the wires for stabilization in the intermaxillary wiring method.

TECHNIQUE FOR THE EXTRAORAL METHOD

The skeletal fixation has been used by the author on a number of cases of pathologic fractures with excellent results. Using the extraoral fixation^{1, 2} as described below, the patient always enjoys a more substantial and appetizing diet postoperatively, due to the fact that more stabilization is afforded by the use of this appliance.

When using the extraoral method, the incision is made and the bone ends rongeuired in the same manner as that in the previously described operation. With a scalpel a superficial stab wound is made through the skin $\frac{1}{4}$ inch beyond the extremity of the incision, and another stab wound is made $\frac{1}{2}$ inch beyond this in a horizontal line; the same procedure is carried out on the opposite side of the incision. These stab wounds should be made on the lateral surface near the lower border of the mandible. The muscle layers are then retracted with blunt Mayo scissors to the depth of the bone. Using a drill point size $\frac{3}{32}$, a hole is drilled at the site of each stab wound through both plates of the mandible. A tapering screw ($\frac{1}{8}$ inch at its greatest width, and $1\frac{1}{2}$ inches in length) is placed into each one of the four holes and screwed into position. The screws

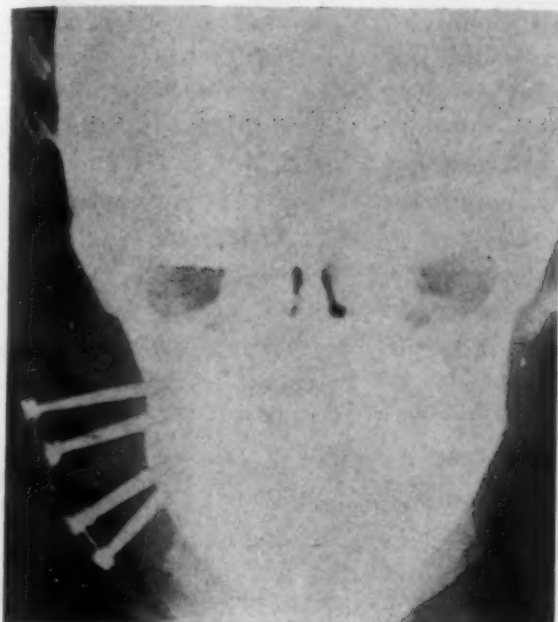


Fig. 7.

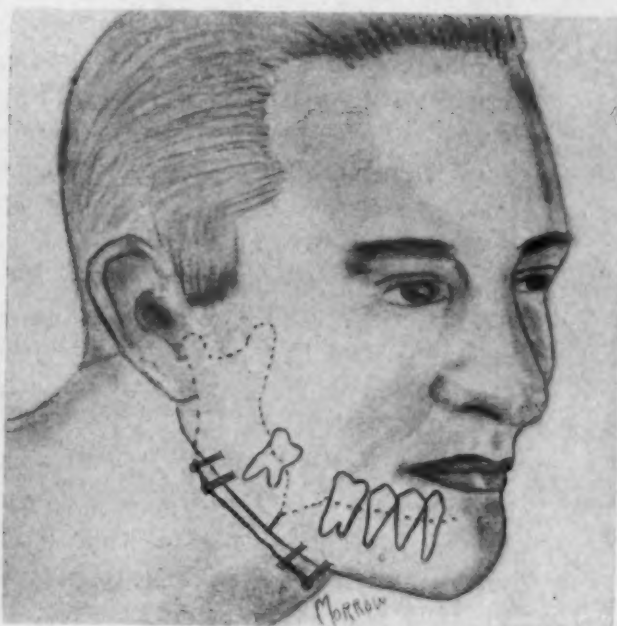


Fig. 8.

Fig. 7.—Radiograph showing the screws in position ready for the application of the plaster splint.

Fig. 8.—Relationship of the wire to the screws, and the screws to the mandible

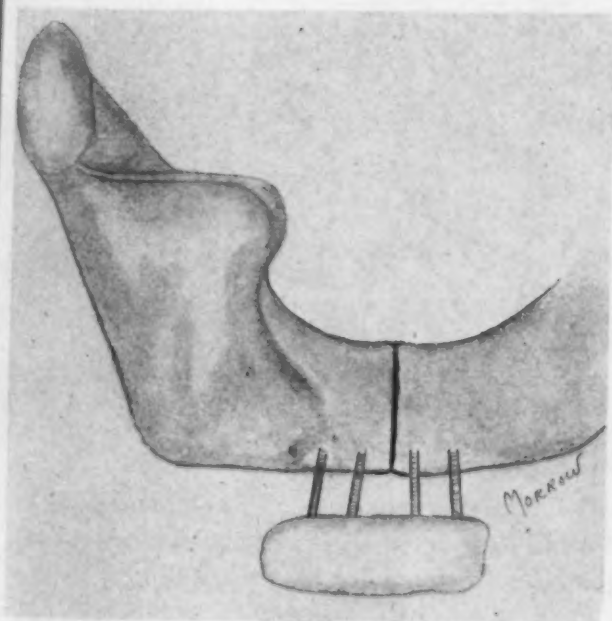


Fig. 9.



Fig. 10.

Fig. 9.—Relationship of the splint to the mandible. The space between the plaster and the bone, in the actual case, is filled with the soft tissues of the face.

Fig. 10.—Relationship of the splint to the face.

must extend completely through the mandible so that the points will be slightly into the musculature of the floor of the mouth; at this level, the screws are below the oral cavity (Fig. 7). It may be mentioned here that so long as the roots of the teeth are avoided, the screws may be placed anywhere in the mandible except above the level of the mandibular foramen where the bone is too thin to afford suitable stabilization. A gauze sponge is placed at the base of each screw to close off any possible contamination. A piece of floral wire is wrapped around the head of each screw to hold it in correct position (Fig. 8). A strip of gauze saturated in a thin mix of plaster is wrapped around the projecting shafts of the two outer screws, thereby enclosing all four screws; approximately four laps are made. The gauze is then pressed together and smoothed down making a cylindrical splint parallel with the lower border of the mandible (Figs. 9 and 10).

TECHNIQUE FOR BONE GRAFTING THE MANDIBLE

For this purpose the most popular, or block bone graft will be described. It is so called simply because it is a block of bone removed from another part of the body, most often from the crest of the ilium, from the tibia, or a rib. Because the tibia is so compact and has such a dense structure that affords little or no passageway for blood vessels, it is not advisable to use a section from this bone.

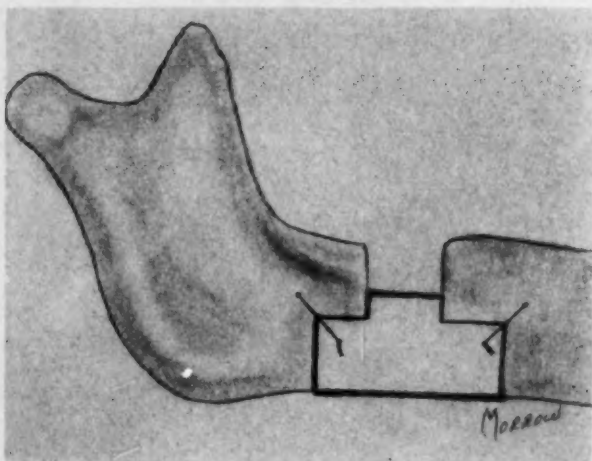


Fig. 11.

Fig. 11.—A method of cutting the graft so that the maximum amount of surface is in contact with the vital bone.

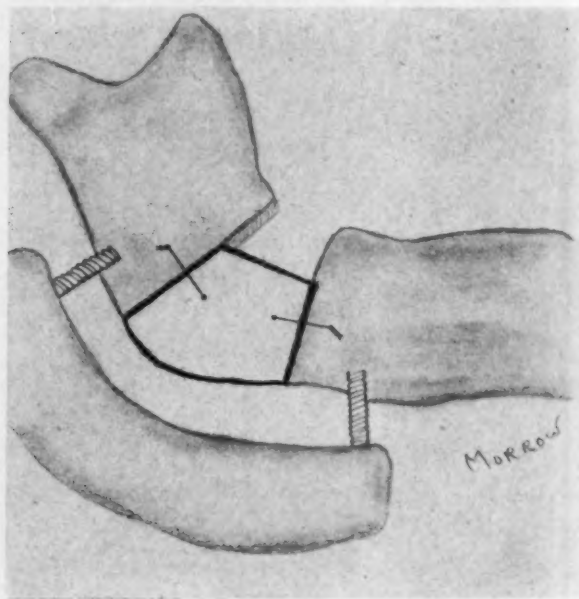


Fig. 12.

Fig. 12.—Bone graft fixation utilizing the plaster splint and silk sutures.

After the tissues have been retracted from the mandible, the bone ends are freshened thoroughly with the bone file or rongeur forceps until spongy bone is reached. At no time should there be a communication with the oral cavity, for this alone is the cause of many failures in bone grafting. The block is cut by an orthopedist, at the time of operation, to fit the measurements of the area to be grafted. The block of bone to be grafted is drilled with holes to approximate corresponding holes drilled in the mandible to receive the fixation by wire or sutures according to the technique to be used. If the wires

are to be used, the graft is then wired to the mandible (Fig. 11) in correct position, and the tissues closed. The wires remain permanently.

If the extraoral fixation is used with the plaster splint, two screws may be placed in the mandibular fragment one on each side of the graft (Fig. 12). The graft is held in position by braided silk, the tissues are closed, the incision covered with gauze, and the splint applied. This leaves the graft with a minimum amount of foreign body. If a third screw is placed through the graft, the silk suture is unnecessary (Fig. 13). The splint must be left in position until there is stabilization proved by radiographs and palpation.

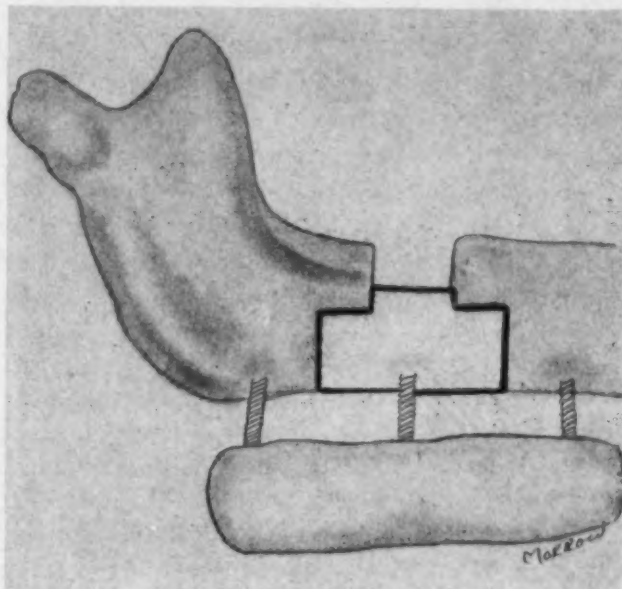


Fig. 13.—Bone graft fixation using the plaster splint with three screws.

CASE HISTORY

The following is an actual case history chosen from a group of several as a typical example of the action of the pathologic fracture.

The patient, a white woman, aged 42 years, presenting a very thin and anemic appearance, was brought to the dental clinic for treatment of a nonunion fracture of three months' duration. The patient also gave a history of active tuberculosis.

History.—Several months ago, the patient for no apparent reason developed an infection of the lower left jaw in the area of the premolars. She sought the advice of her doctor. The radiograph showed involvement of the mandibular bone which was apparently osteomyelitic in nature. She was kept on hot compresses. The jaw continued to drain and the condition became progressively worse. Upon one of her visits for treatment, it was discovered that the mandible was completely fractured in the area of infection. A sequestrum was removed, and the teeth were wired by the intermaxillary method. This wiring remained for three months, and at this point the patient entered the dental clinic for treatment.

Radiographs were taken and the bone extremities were found to be rounded, but approximated to a fair degree. There was no union and no remaining infection.

Operation.—The tissues in the mouth were healed, leaving no opening. Therefore, under intratracheal anesthesia using cyclopropane, a small incision was made along the lower border of the jaw, and the tissues were retracted from the bone. The operative procedure used was the same as that described previously for open reduction using wire.

Progress.—The patient was ordered to have no solid foods for five weeks. She was discharged from the hospital on the fourth postoperative day, at which time the sutures were removed. She was given an appointment to return to the clinic once a week for checkups.

The eighth postoperative week, more radiographs were taken, and it was found that there was no infection nor resorption of bone. There was union, and at this time the patient could exert a considerable amount of pressure during mastication. The wire was left in position, and the patient discharged with no complaints.

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PROLONGED RETENTION OF THE DECIDUOUS TEETH

CASE REPORT

JULIOUS R. BOURGOYNE, B.S., D.D.S.,* AND FAUSTIN N. WEBER, M.S., D.D.S.†

A REPORT is often circulated that a patient was found and observed entertaining the growth of a third set of teeth. I am quite sure that it may be said without fear of contradiction that most of these statements are erroneously distributed. However, it is the true belief of the observers, in most cases, that the patient is actually getting a third dentition. Probably no one had dealt with accurate records on these patients; therefore, when the dentist was consulted, the story was related to him and the idea was immediately created concerning three complete sets of teeth.

I would like to offer here the brief case history of a patient which will present the typical basis of the ideas behind such rumors.

It was found that the patient received his complete deciduous dentition, and in due time lost some of them, which were replaced by the permanent teeth. After studying the case, however, it was also found that the patient now at the age of 29 years has failed as yet to lose the remainder of these deciduous teeth, the shedding of which is long overdue. Radiographs were made and several permanent teeth were found embedded and impacted within the arch. The patient was greatly pleased to find that he was not really a freak but simply the victim of unfortunate circumstances which had developed early in life.

It is my firm belief that the majority of these third dentition cases are of such a nature as that presented here, and if the complete history, as nearly as possible, could be obtained, each case would show a similarity.

CASE HISTORY

Mr. C. R. was born one month prematurely of a 16-year-old mother. His weight was not recorded at the time of birth, but at the age of 5 months he weighed 3½ pounds and remained under the constant care of a physician for a period of two years. His weight remained low and at the age of 15 years he weighed only 75 pounds, but thereafter began to gain.

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Mr. C. R. is now the largest of nine children in the family, even though he was the only one of the group abnormal at birth. He is 6 feet tall, weighing 195 pounds.

He has had "weak eyes" since the age of 18 years and now suffers occasionally from ulcers of the stomach.

There was no known serious sickness in his family as far back as could be traced. He was of a poor family, and lived in the country most of his early life.

The one child of Mr. C. R. was examined and in every respect seemed to possess a normal mouth.

It has been noted on several occasions by this writer that children who were born prematurely, or at birth were exceedingly small, entertain deformities of the dental arches, the teeth, or their relations.

It is interesting to note also that there are more deformities in the teeth and their positions in the arches, than in any other organ or part of the body. Even the dentist who has studied teeth is very eager to remark on the beauty of teeth that are good and in correct alignment, yet remarks are seldom made concerning someone who has all of his fingers present and in correct position. It seems that we naturally expect this. On the other hand it seems that it is always the "abnormal" that we expect when examining the mouth.

In many cases we cannot prevent the malposition of teeth, but, in order that children may enjoy more nearly "normal" occlusion and position of the teeth and their surrounding structure later, care should be exercised to diagnose and treat these cases early.

Detailed observations of this case are given by F. N. Weber in the following paragraphs.

Examination.—An intraoral examination revealed the presence of the following teeth (Fig. 2):

$$\frac{76ede21}{76e4cb1} / \frac{12ede678}{1bc45678}$$

The dentofacial anomaly was classified as a Class I Angle, Class II Bennett type of malocclusion characterized by an extremely deep vertical overbite and complicated by multiple retention of primary teeth. $1/1$ were in linguotorsion; $2/2$ were in labiotorsion. The mandibular arch was constricted in its anterior segment with slight crowding evident in the mixed dentition present in that area. Marked abrasion of the primary teeth was noted. What appeared to be abrasion caused by faulty toothbrushing methods was found in the cervical third of labial aspect of c/e .

Full-mouth roentgenograms revealed the presence of all permanent teeth, except $\overline{8}$, with succedaneous teeth unerupted for those primary units of the dentition that had not been exfoliated, i.e., $\frac{ede/cde}{ecb/bc}$. (Fig. 1, A, B, and C).

The crowns of all unerupted permanent teeth appeared to possess normal form; their roots were slightly shortened. The majority of the unerupted permanent teeth were ectopically related to their primary predecessors. $543/345$ were in mesial axial perversion. $2/2$ was in distal axial perversion.

The roots of all retained primary teeth exhibited resorption. This was especially pronounced on b/c . Pressure upon the roots of the retained primary teeth did not appear to be a factor in their resorption since the crowns of most of the unerupted permanent teeth were some distance from the resorbed roots.

There was interproximal caries on $e/$ and rarefaction of the alveolar crest throughout the dentition in the interproximal areas. Some serusal calculus was evident on $1/1$.

An extraoral examination revealed all the facies of a dentofacial anomaly characterized by a deep vertical overbite. The lower third of the face was lacking in vertical height when compared to the middle and upper thirds. A deep labiomental sulcus was present and there was slight eversion of the lower lip.

Diagnosis.—A tentative diagnosis of multiple ankylosis of primary teeth was listed. The manner in which the clinical signs confirmed this diagnosis was the relative immobility



Fig. 1.

of the primary teeth; it denied the diagnosis in the fact that the occlusal plane of all the primary teeth, presumed to be ankylosed, placed them in functional contact with opposing primary and permanent teeth. Ankylosed primary teeth are usually out of functional contact with teeth in the opposing dental arch, especially so if the ankylosis has been of some duration. This has been explained by the fixed vertical position of the ankylosed tooth and the subsequent continued eruption of the other dental units with the coincident vertical growth of their supporting alveolar processes. Both growth mechanisms literally wedge the jaws apart, leaving the ankylosed tooth short of the occlusal plane.



Fig. 2.

It may be that in this case the large number of primary teeth, present and ankylosed, tempered the vertical growth forces sufficiently to allow the teeth to remain in functional contact. It is more likely that some general factor or factors are concerned in the retention of the primary teeth. The fact that there is no near contact of crowns of the succedaneous teeth with the roots of their primary predecessors suggests that retained teeth are not a factor in the noneruption of the permanent teeth. In this aspect the case differed from the roentgenographic findings of ankylosis.

In such doubtful cases a positive diagnosis of ankylosis can be made by surgically removing the primary teeth with a portion of their supporting alveolar process. These specimens are frozen, sectioned, and stained according to the technique suggested by Cahn. Microscopic examination of the sections so prepared makes positive diagnosis possible.

Treatment.—Other than the laboratory examinations recommended for the purpose of diagnosis and to ascertain what general factors are concerned in the condition, extraction of all deciduous teeth was recommended. If the retention of the primary teeth is not a cause of the noneruption of the succedaneous teeth—and such appears to be the case—it is unlikely that the permanent teeth will erupt solely because the primary teeth are extracted.

Prognosis.—The prognosis is doubtful, lacking specific information of the etiology; the treatment empirical for the same reason.

Comment.—It has not been possible to follow this case through to completion because the patient left for the Armed Forces shortly after the appointment made for examination.

PROSTHETIC RESTORATION WITH A REMOVABLE PARTIAL
DENTURE AFTER SURGICAL REMOVAL OF A TUMOR OF THE
RIGHT MAXILLARY ANTRUM

CASE REPORT

JAMES M. STRICKLAND, D.D.S.*

SURGICAL toll in the removal of tumors often necessitates a certain disregard for the esthetic quality of the patients' appearance. The prosthodontist serves mankind well in the restoring of these unfortunately lost features. The art of constructing artificial eyes, ears, and noses is indeed an enviable specialty. As important, however, to complete esthetics is the art of restoring the dentition and the facial contours supported by the teeth.

Fig. 1.

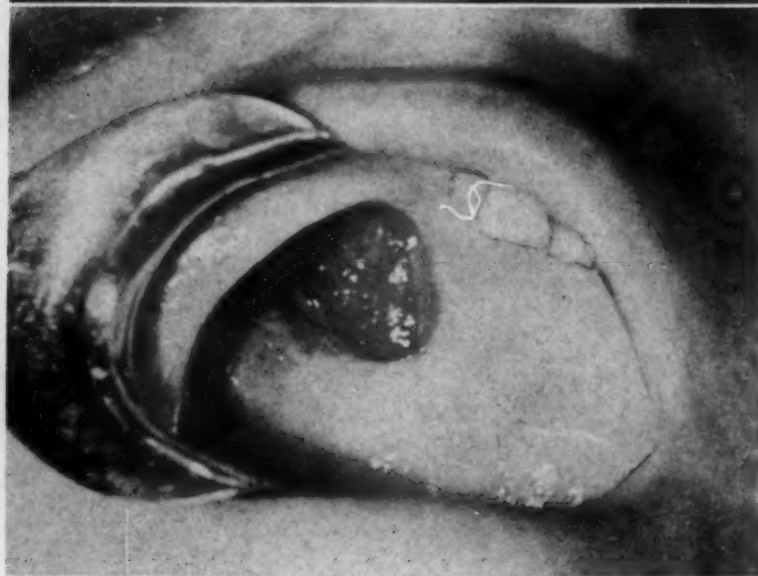


Fig. 2.

Fig. 1.—Extraoral esthetics. Note depression of right half of upper lip and right cheek.

Fig. 2.—Intraoral esthetics. Palatal opening enters into the nasal cavity, nasopharynx, ethmoids, and orbit.

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Patient R. C., aged 18 years, presented to the prosthetic clinic external esthetics as shown in Fig. 1 and intraoral esthetics as shown in Fig. 2.

The patient's medical history* reveals that roentgenographic examination on April 27, 1942, indicated "an opacity of the right antrum which is apparently an expanding soft tissue growth. The floor of the orbit is pushed upward, the anterior walls outward, the floor of the antrum downward, and the nasal wall into the nares. At no point is any erosion visible. Dental films show no evidence of a dentigerous cyst or adamantinoma. The right molars and premolars are displaced by the growth. Since no erosion is visible, it is believed that this is either a rare type of sarcoma of the antrum or a large mucocele."



Fig. 3.



Fig. 4.

Fig. 3.—Labiobuccal view of the gross specimen, the right superior maxilla.

Fig. 4.—Sagittal view of the gross specimen.

On May 5, 1942, he was admitted to the Baptist Memorial Hospital, Memphis, Tennessee, with the complaint that he had noticed, a year before admission, a swelling of the right maxillary region and a decrease in the ability to breathe through the right nostril. The growth was rapid. Preliminary examination revealed no increase in temperature, no tenderness, and no nasal discharge. His ears were negative. There was no pain in the enlarged mass. Family history was essentially negative. Past history indicated usual childhood diseases, typhoid fever at 5 years, and no previous operations. Physical examination indicated nutrition good, eyes and ears negative, right side of nose completely blocked by deviation of lateral wall, septum straight, right maxilla showing a 2+ prominence, no tenderness; right half of roof of mouth slightly lower than the left side, no tenderness; the entire right side of face considerably larger than the left side. Heart, lungs, and abdomen were negative. Blood examination revealed Wassermann test negative; hemoglobin, 82 per cent (13.9 Gm.); erythrocytes, 4,460,000 per cubic millimeter; leucocytes, 5,850 per cubic millimeter; small lymphocytes, 34; large mononuclear lymphocytes, 1; neutrophils, 64; juvenile cells, 3; stab cells, 6; segmented neutrophils, 55; eosinophiles, 1.

Roentgenographic report, June 3, 1942: "Examination of the right antrum after introduction of iodized oil into the cavity of the antrum shows that the antrum itself has been

*Obtained through the courtesy of the Baptist Memorial Hospital and Dr. W. Likely Simpson.



Fig. 5.—Intraoral roentgenographs. Right maxillary canine, premolar, and molar views taken of gross specimen.

pushed laterally and that the place usually occupied by the antrum is now occupied by a large soft-tissue tumor. This tumor is somewhat cystic in type and apparently arises from around the roots of the cuspid or eye tooth. We believe it is most likely some form of dentigerous cyst."

Biopsy report, June 8, 1942: "Specimen consists of several fragments of yellowish-white tissue. Permanent section is composed of bony trabeculae interspersed with dense fibroblastic structure. There is apparently no evidence of malignancy. Request: another biopsy."

An exploratory operation and biopsy were done on July 21, 1942. "An incision was made over the right antrum between the gum and upper lip. Bone of the anterior wall of the antrum was removed from the lower margin up to the infraorbital ridge. The floor of the antrum was curetted. The incision was left open." A specimen was sent to the laboratory for biopsy. Gross findings at this time: "Anterior wall of right antrum replaced by tumor which consisted of a cystic spongy thick base about $\frac{1}{3}$ inch in diameter which had invaded the nasal bones and hard palate." Operative diagnosis: "Adamantinoma of right antrum."

The patient was discharged on July 26, 1942, in "good condition." Final diagnosis: "Adamantinoma of the right antrum."

On Aug. 16, 1944, the patient was readmitted to the hospital. Roentgenographic examination on Aug. 17, 1944, reports: "Re-examination of the right maxilla shows that the large bony mass which has been previously demonstrated has increased in size. It apparently arises from the roots of the right upper cuspid or canine and fills the antrum on the mesial side displacing the cavity of the antrum laterally. It is considerably larger than the original film showed. We still believe that it is most likely a large dentigerous cyst."

On Aug. 18, 1944, a modified Moure operation on the upper right jaw was done by Dr. W. Likely Simpson, surgeon, of Memphis, Tennessee. "An incision was made along the right side of the nose and below the eyelid. All soft tissue was removed from the anterior surface of the maxilla. The hard palate was split with a chisel and a cut made through the malar process and frontal process of the superior maxilla. The entire mass was removed just anterior to its attachment to the pterygoids. The skin was closed with fine silk and the wound packed with iodoform gauze."

The laboratory report of Aug. 18, 1944, was as follows: "The specimen consists of the right half of the superior right maxillary bone. The antrum is filled and practically the entire bone structure has been replaced by a tumor tissue which has the appearance of cancellous bone." (Figs. 3 and 4.)

"Permanent section: The sections are composed of trabeculae of bone in which the cancellous spaces contain immature fibrous tissue. Rows of osteoblasts are found along some of the trabeculae. Diagnosis: Benign fibrous osteoma."

Final diagnosis at the time of the patient's discharge, Sept. 1, 1944, was adamantinoma.

Intraoral roentgenographic examination before prosthetic restoration revealed the remaining teeth and bone are unusually healthy (Fig. 5). The roentgenographs of the maxillary right quadrant were made from the gross specimen that was removed. There seems to have been no third molar tooth in this quadrant.

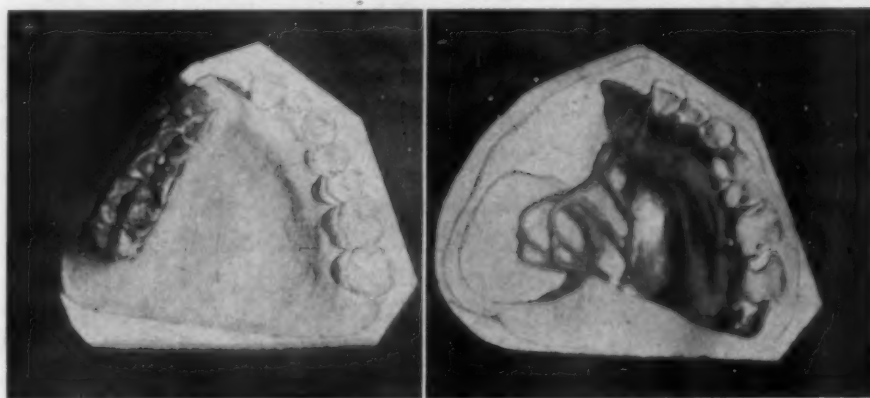


Fig. 6.—Models with bite rim and unfinished casting in place.

Fig. 7.

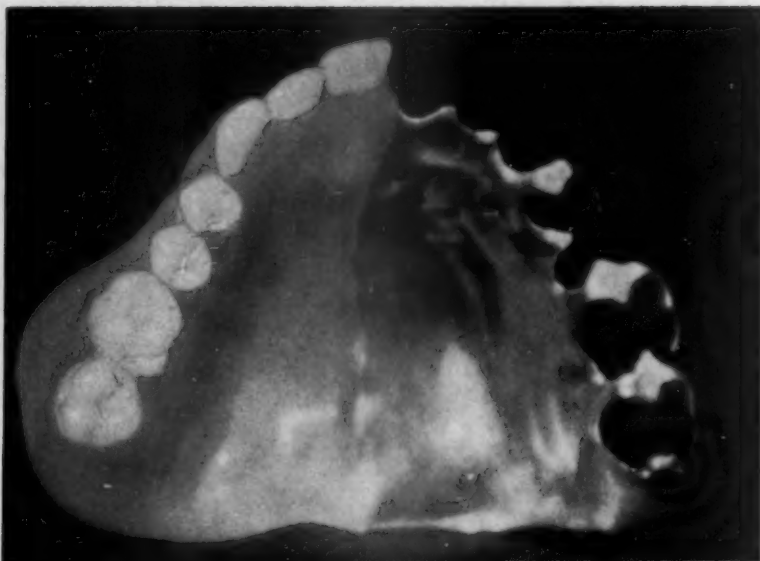


Fig. 8.

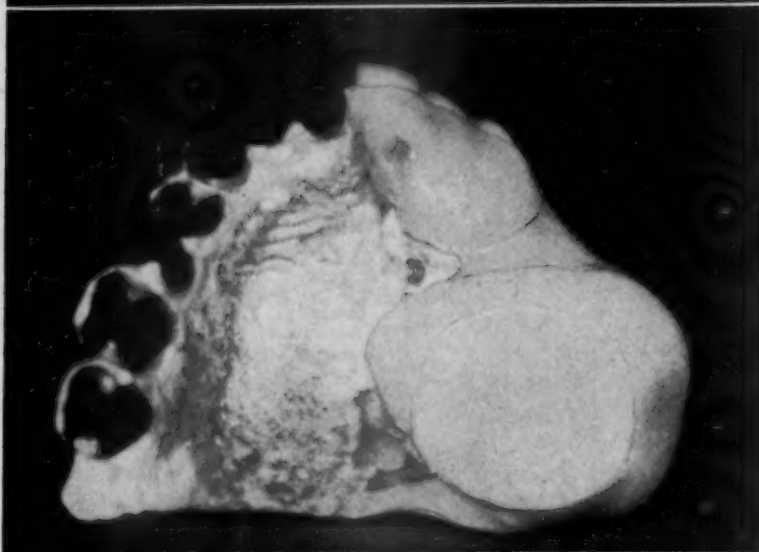


Fig. 9.



Fig. 7.—Occlusal view of prosthetic restoration.

Fig. 8.—Palatal view of prosthetic restoration showing the acrylic extension into the palatal opening and buccal contour for support of the collapsed cheek.

Fig. 9.—Labio-buccal view of the prosthetic restoration.

TECHNICAL PROSTHETIC DIFFICULTIES

The most obvious difficulty in the restoration of this masticatory and esthetic mechanism is the stabilization of the removable partial denture. (Figs. 6 to 9.) The available undercut (retentive) areas for clasp retention are at a minimum, due to the anatomic shapes of the crowns of the teeth. It can be seen on the models that the crest of convexity of most of the axial tooth surfaces is at or very close to the gingival crest. The key retentive areas are the mesial surface of the left maxillary central incisor, the distolabial surface of the left canine, and the distobuccal surfaces of the first and second molars into which areas clasps were designed.

Fig. 10.

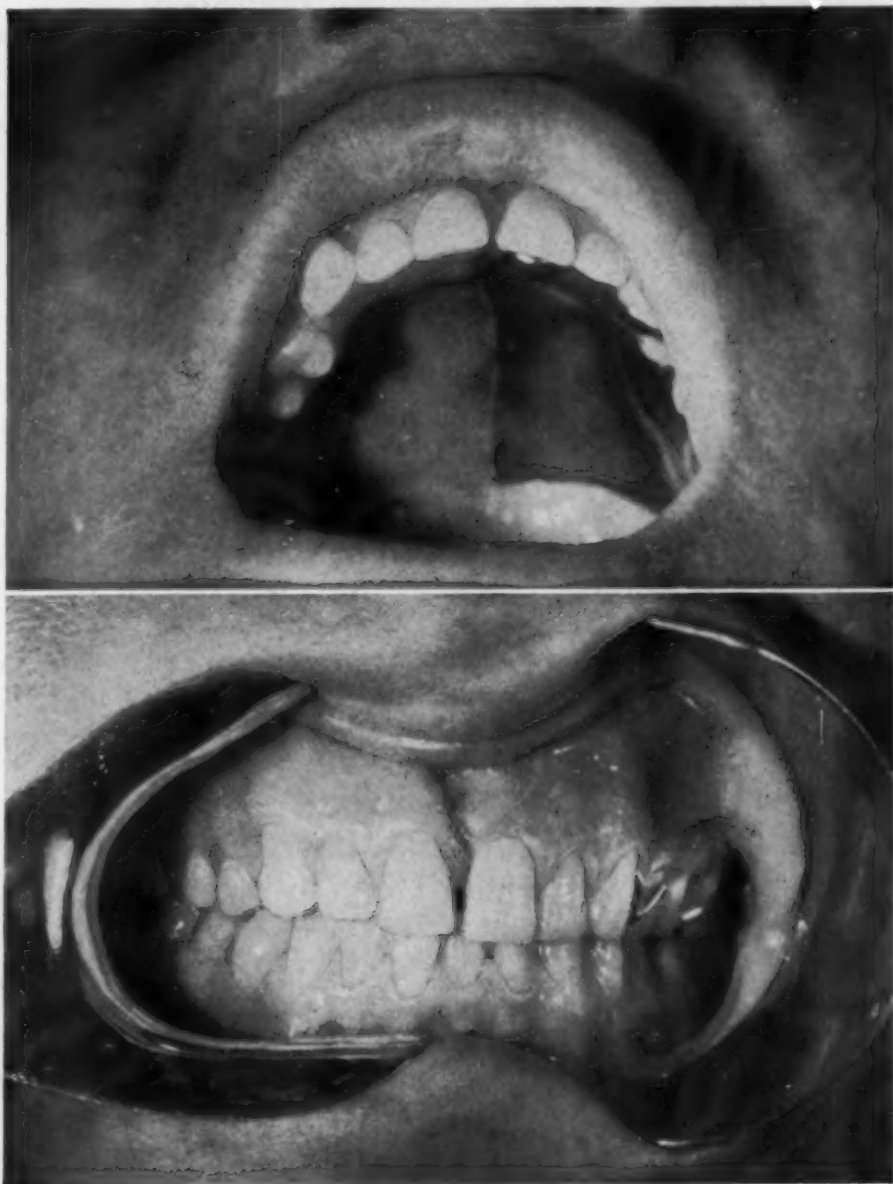


Fig. 11.

Figs. 10 and 11.—Prosthetic restoration in place.

Bracing action is secured by adapting the casting to the lingual surfaces of all teeth, the buccal surfaces of all claspel teeth, and the contact of the palatal plate and acrylic extension into the palatal opening with the surrounding mucosa.

Support is adequate due to palatal and tooth rests and buccal clasps. Obviously this is less over the palatal opening on the right than on the left, but esthetics and sufficient masticatory efficiency are secured.

For proper support of the right cheek by the acrylic base and teeth an individual impression tray was made with impression compound to overextend the denture area and secure tissue compression over the palatal support area. This impression was relieved sufficiently around the axial surfaces of the teeth to allow an adequate thickness of corrective material and a final impression was made using an alginate paste for tooth and tissue detail.

The patient's left maxillary central incisor presented a transverse band of cloudy enamel in the incisal half of the crown. This was duplicated on the right prosthetic central incisor by staining of the porcelain. These are not clearly visible in the photographs.

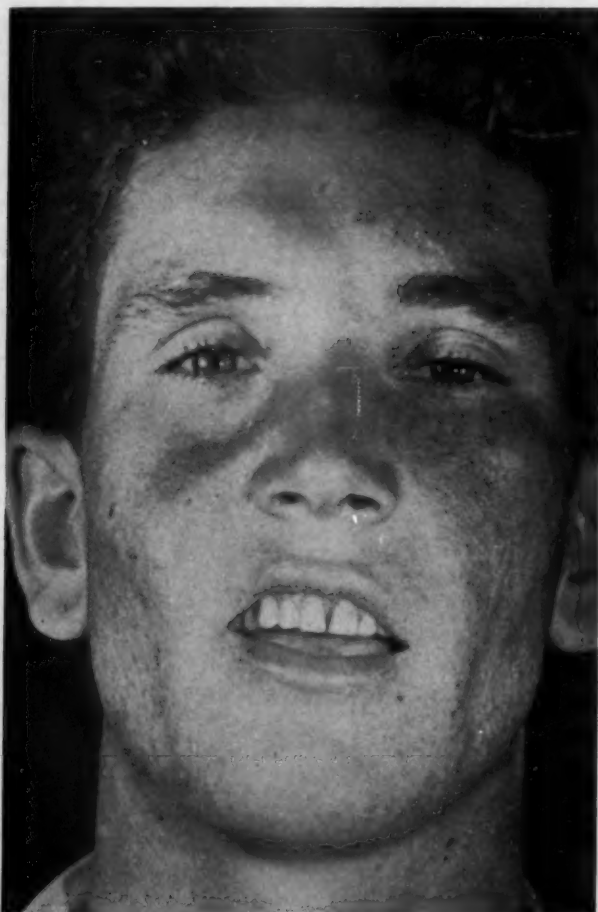


Fig. 12.—External esthetics. Note depressed infraorbital ridge that will be supported by a future bone graft.

The first casting of ticonium fit the master model but did not fit the mouth well enough, thus indicating an inaccurate master impression. This casting is shown in Fig. 6. A second impression was made with the impression compound and alginate paste and a second casting made that fit well, with surprising retention.

A tight elastic band of fibrous tissue in the right cheek complicated the supporting of the cheek by the base material. When it was supported adequately for esthetics, it had a strong tendency to unseat the removable partial denture on the right side where retention is at a minimum. By trimming the waxed case to fit this band of tissue, satisfactory esthetics was obtained. The possibility of this tissue further contracting in the future and attempting more to unseat the appliance was not disregarded, but observation is the only criterion for this event.

The patient has worn this removable partial denture for six months without difficulty, reporting that he "often forgets about it until mealtime" (Figs. 10, 11, and 12).

SURGICAL PREPARATION OF THE MOUTH FOR ARTIFICIAL RESTORATION

LOYD CLAYTON TEMPLETON, D.D.S.*

IN THE past there was a tendency in dental practice to put surgery and restorative prosthetics into separate categories. The prosthodontists were forced to work with the mouth as the surgeon had left it. Too much dependence was placed on Nature when the work could have been done more satisfactorily and quickly by the surgeon. Mouths were left for Nature to prepare for dentures because of lack of surgical skill, poor cooperation between surgeon and prosthodontist, fear of losing a prosthetic patient by suggesting surgery, failure of explaining to the patient the necessity for the correction, and fear that remunerative compensation would not cover time spent doing surgery. In making corrections of abnormal conditions in mouths preparatory for artificial restorations, I have found results to be exceptionally gratifying.

The rapid development of dentistry now challenges its operators of today to know and practice surgical preparation of the mouth. They must study their cases from a cosmetic as well as a restorative viewpoint. The professional reputation and character may be either enhanced or curtailed by the standard of their surgical operative technique, their psychological treatment of their cases, or the atmosphere created by their office appearance and personnel. The public's increasing knowledge of asepsis, and improvements in dental equipment, dental training, and anesthesia, have produced an observing and critical individual. We have a more obligatory relationship to our patient with each advancement of dentistry.

As a practitioner, one must realize these facts and dispel any fears that may be relative to this subject of surgical preparation of the mouth. Answer the following questions, and then, if indicated, proceed with the work, knowing that improvement of an undesirable situation in favor of a more satisfactory restoration is being accomplished: When and why is surgical preparation indicated? Will the results be an improvement in the particular case at hand, or in the majority of cases? The first question will be answered in the discussion of the oral examination. The answer to the second question is found to be evident through this article.

The patient must be educated to the necessity of surgical correction. The mistakes of the past are far too prevalent today in the offices of men unwilling to progress with the profession, who, rather than help, degrade and hinder further progress of this study. Surgical correction of the mouth is now an accepted part of ethical practice. It is the responsibility and obligation of every practitioner to educate both himself and his patients to this corrective work. A clear explanation to the patient usually dispels fears and stimulates submission to the surgery.

The first principle of surgery to be considered is the adequacy of the surgical judgment and skill of the operator, which no one knows better than

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the operator himself. "Let the eye go before the hand, and the mind before the eye."

Rapid development of prosthetic dentistry demands the closest cooperation of the prosthodontist and oral surgeon as a means of discovering and correcting conditions in the oral mechanism—the correction of which renders the prosthetic management of the cases less difficult. The surgeon should be guided by the recommendations of the prosthodontist.

Conservative, yet adequate, process trimming and soft tissue correction are of great importance. If too much bone and soft tissue are removed, inadequate retention is the result, whereas too little removal will bring about lack of correct adaptation. This conservation is the result of preoperative visualization of the healed corrected ridge and a planned operative procedure to attain the desired results with minimum trauma and relief, thus permitting the processes of bone resorption and regeneration to follow normal courses. Bone surgery also necessitates surgery of the soft tissues. Operations are successful only when treatment of both is in harmony with proper surgical principles.

No part of the human body permits the surgical abuse that is tolerated by the oral tissues. Too often are seen the mutilated cases where the predominating thought was tooth removal, trusting to the kindness of Nature for a protecting influence acting like a magic wand to heal and correct all of the poor surgery done and work left undone. Due to the tolerance of the oral tissues and the high degree of immunity to fatal results from oral infections, we receive this protection to a remarkable extent. This is fortunate as it saves many lives after poor surgery, but unfortunate as too many operators are not required and, therefore, not stimulated to learn and practice correct surgical technique.

In process trimming the undercuts must be removed as completely as the case will permit, but, since the periosteum and gum tissues do not offer adequate support as a matrix for bone regeneration in contouring ridges, there must be left sufficient alveolar process to form the matrix. Careless loss of an excessive amount of the investing tissues in the removal of teeth increases the difficulty of securing good results in full mouth preparation. Proper operative technique requires the preservation of all the tissues of the dental ridge, except those portions best removed at the time of the extraction to prevent loss of more extensive amounts due to trauma.

In both the mandibular and maxillary canine regions, the labial plate forming the canine eminence may be fractured during the extraction of these teeth. When this occurs, more bone than just the part forming the eminence is lost, usually one-third to one-half of the mesial and distal septa, thus creating an ugly ragged depression that renders proper denture construction more difficult. When this fracture is anticipated or feared, proper steps should be taken to prevent its occurrence. Prevention consists merely of conservative but adequate relief of the labial plate to the extent of removing the undercut created by its prominence. The tooth may then be extracted without process fracture and undue trauma, thus leaving the septa to support the mucoperiosteum and prevent an undesirable depression.

Anatomic knowledge of the structures to be operated on and the surrounding structures is an essential surgical principle. Detailed anatomy may and should be reviewed often from textbooks. A few essential points relative

to this subject are here presented. The relation of various parts one to another, and arrangement and strength of bony plates in various locations are necessary for an intelligent procedure. Both the mandible and maxilla consist of an outer and inner plate of hard cortical bone, the space between being filled with rather soft cancellous bone. The cortical bone is subperiosteal and typical Haversian system bone. The cancellous bone is arranged as trabeculae, extending in such directions as best provides required strength. The intertrabecular spaces contain marrow. The lingual cortical plate usually is thicker than the labial, but is weaker on the mandible, due to lack of support. A thin, somewhat less dense and more porous layer of bone lines each alveolar socket, and is called the lamina dura. The labial and lingual cortical plates are well utilized in the removal of teeth with elevators, using these plates as points of application of force and fulera for leverage.

The various foramina of the maxilla and mandible are important structures in surgery of these bones. The one most often encountered is the incisive foramen. When considerable process must be removed from the anterior segment, this foramen, through which pass the nasopalatine nerve and vessels, approaches the crest of the ridge where pressure from a denture will cause discomfort. Relief to that extent should be avoided when possible. Otherwise, injury to the structures in this area should be avoided, and adequate relief of the denture over this area is essential.

Muscle attachments should not be disturbed in the average case. Muscular control of artificial dentures is decreased if these attachments are destroyed. Only when a muscle definitely interferes with proper denture construction, an unusual situation, should its attachment be moved.

The malar process of the maxillary bone presents on its inferior surface a ridge known as the "key ridge," because it usually locates the first molar tooth. When the base of this process extends over the buccal roots of the molars, the difficulty of extraction without fracture is greatly increased.

The posterior end of the maxillary alveolar process constitutes the tuberosity and consists mostly of spongy bone. When extracting third molars, especially those with marked distally inclined roots, this tuberosity may be fractured with a resulting flattening of the ridge—a very undesirable condition in denture construction.

The maxillary sinus situated in the body of the maxillary bone presents its floor in rather close relationship to the apices of the roots of the first and second molars, and occasionally the second premolar and third molar where the intervening bone is very thin or weakened by infection. Fractured roots are often forced into the antrum when removing them.

The mandibular vessels and nerve pass beneath the roots of the mandibular teeth and may be injured during extraction of a third molar or curettement of an infected area at the apex of a tooth. Beneath, and usually between the root apices of the first and second premolars is situated the mental foramen, through which pass the mental vessels and nerve supplying the mucous membrane and integument of the lower lip and chin. Injury to either the mental or inferior dental nerves may result in paresthesia or severe pain.

In the third molar region the anterior border of the ramus is sometimes overlying the buccal plate. This may be either an advantage or a detriment. The ramus may often be used as a fulcrum in applying the force with an elevator to dislodge the third molar; however, if it becomes necessary to remove the buccal plate, the heavy bone will, of course, complicate the procedure.

While the alveolar process suffers some resorption as age advances, it also becomes more dense, less springy, and less porous. The periodontal membrane seems to be thinner, and in certain areas on the root to disappear entirely. Clinically, the teeth seem to become more brittle. Whether or not these changes actually occur, experience teaches that in extracting teeth of older people, the lateral movements must be done more cautiously.

The alveolar process has always been regarded a transitional tissue, but there is good reason to believe that if adequate stimulation is provided after extraction of the teeth, the alveolar process will not only remain, but it will, also, fortify itself by the growth of new bone to withstand such strains as may be placed upon it. The disappearance of the alveolar process is most noticeable where teeth have been replaced by extensive bridgework, stimulation from usage being absent.

When stimulation of pressure in chewing is removed and all the stress borne by the bridges, the alveolar process gradually resorbs until entirely gone. The absence of a ridge in the mouths of patients who have worn large bridges supported by few teeth becomes quite evident when the remaining teeth are lost, and the prosthodontist is faced with a difficult problem of restoration.

Preservation of the alveolar process, on the other hand, is well illustrated in those patients who have either gone without dentures and have become accustomed to chewing on the ridges, or who have had dentures that so distributed the pressure as to stimulate the tissues and keep the ridges firm and hard. It has been shown by Noyes that after extraction of a tooth the socket fills up first with connective tissue, the growth of bone continuing until this is completely replaced by bone. Whether this bone is retained depends upon the use of it—the integrity of this new bone being governed by Wolfe's law, stating that bone is formed of such size and strength as to withstand the strains of its intended use. MacMillian, therefore, urges the chewing of vegetables and paraffin by the patient as soon as possible.

The most ideal ridge form for the reception of dentures is one from the widest labiolingual portion of whose base the ridge tapers without interference toward the crest. The reasons for this are easily explainable and understandable. Take, for example, an untrimmed ridge with a labial undercut, this undercut portion being wider labiolingually than the base of the ridge: the pressure (load) of the denture is supported only by the crest of the ridge which will resorb rapidly, thus allowing the denture to settle. The labial adaptation and peripheral seal are then broken, resulting in loss of retention. The prepared (trimmed) ridge, however, bears the load over the entire ridge, thus resorbing uniformly and more slowly, the settling denture becoming more firmly seated and increasing retention. Since retention is so desirable a feature in denture construction and so difficult to obtain, it seems reasonable that time, effort, and expense of proper ridge preparation increasing retention are well spent.

ORAL EXAMINATION

In the planning of surgical correction, the entire oral cavity should be examined in an attempt to discover the presence of any indications of systemic diseases. A good criterion to follow is that of an old retired practitioner, who said: "The first thing I look for in a person's mouth is syphilis." If one is careful enough to eliminate this "mimic of all diseases," he can be fairly certain that he has a healthy mouth with which to work.

Evidence of acute gingival irritations and degeneration indicates immediate bacteriologic examination to eliminate Vincent's infection—the presence of which indicates postponement of surgical procedure.

The presence of abnormal growths should be investigated as to their origin and history to determine the possibility of malignancy. If a diagnosis cannot be made by the clinical findings and history, a biopsy is indicated.

The usual features should be looked for in the mouth that is to receive dentures. Tissue tone is as important as any factor when planning the mouth for dentures. The oral tissues respond very readily to the activity of the systemic system. We believe that all patients are benefited, and operative prognosis made more favorable when a systemic treatment is instituted that will stimulate the organs of secretion and excretion.

The following prescription is recommended for a preoperative reconditioning of the oral tissues:

| | |
|--|----------|
| Sodium acetate | 30 Gm. |
| Sodium thiosulfate | 30 Gm. |
| Flavor as desired | |
| Water, in sufficient quantity to make | 130 c.c. |
| Take teaspoonful in one-half glass of water before meals for a week. | |

It is often wise to continue taking this preparation for some time after the operation, depending upon the amount of organic stimulation.

It is also important that the patient have a well-balanced nutritious diet, containing fruit juices, milk, fresh fruit, raw and cooked vegetables.

Prophylaxis and medication of the tissues are necessary before operation.

A few years ago it was considered advisable to dismiss a patient for a few months after extraction of teeth to allow healing and resorption of the process to take place before dental construction. Many of these cases returned with ridges satisfactory for prosthetic work. A great number returned with unfavorable conditions, such as:

1. Prominent upper anterior ridge.
2. Prominent buccal plates.
3. Excessively large tuberosities.
4. Torus mandibularis lingual to canine, premolar, and molar teeth.
5. Prominent knife-edge mylohyoid ridge.
6. Torus palatinus.
7. Labial or lingual frena attached too high on the ridge.
8. Muscle attachments too high.
9. Flabby soft-tissue ridge.

When any or all of these conditions have been seen, a set of full mouth radiographs should be taken, even if the mouth is supposed to be edentulous. Too many dentures are constructed on ridges containing root tips, residual areas of infection, unerupted third molars, impacted canines, and the summits of ridges which are too irregular and composed of sharp projections of bone that will be constant sources of irritation under the denture.

ANESTHESIA

With the corrections planned and submission to surgical procedure established, the choice of anesthesia is the next consideration. In the majority of cases local anesthesia is satisfactory. It relieves the patient of a great part

of the worry usually induced by the thought of a general anesthetic. Operating time is lengthened and not so limited to one sitting as in general anesthesia, permitting more skillful surgery with minimum trauma and postoperative discomfort. Local anesthesia reduces hemorrhage and eliminates hospitalization. We use the subperiosteal injection rather than the block method, using a 2 per cent solution of novocain with a high epinephrine content. Small injections of solution are advised, causing the least distortion to soft tissues.

OPERATIVE SURGICAL PROCEDURES

Areas needing correction may be classified as follows, and the corrective procedures are discussed according to this outline:

A. Undercuts Due to.—

1. *Prominent Maxillary or Mandibular Anterior Ridge.*—This condition must be corrected to allow sufficient room for the labial plate of the denture without protruding the upper lip; to reduce the labial undercut to facilitate taking the impression; to reduce irritation of insertion and removal of the denture; to prevent breaking the peripheral seal and labial adaptation when the denture settles. These points are very important in determining the life and success of full dentures. When mucoperiosteal flaps are turned in the removal of bony prominences, the soft tissues should be retracted sufficiently so that they will not be traumatized, and to afford a clear view of the area to be removed. After removal of the bone, there is usually an excess of soft tissue which is trimmed closely, approximating that on the opposite side of the ridge, and this can be adapted and sutured.

A prominent anterior ridge segment may be due to abnormal union in the midline, exostosis due to traumatic occlusion, or a naturally protruding arch. Whatever the cause, the undercut must be removed. A single crescent-shaped incision over the crest of the ridge, extending across and slightly beyond the portion to be removed, usually suffices. Should more access be necessary, the most posterior limit of the incision may be extended cervically and posteriorly about 5 to 10 mm. as required. With the undercut clearly exposed, the excess bone is easily chiseled away and filed smoothly. Necessary care must be taken not to relieve into the anterior palatine foramen, unless in exceptional cases of severe facial deformity. Excessive hemorrhage may result from cutting of the anterior palatine vessels. If direct pressure fails to arrest the bleeding, a wooden plug may be adapted and driven directly into the foramen.

2. *Prominent Buccal Plates.*—When the crest of the buccal alveolar plate is prominent enough to create an undercut, it must be removed for relief. This can be done more easily and with less trauma at the time of extraction of teeth when turning the mucoperiosteum away from the bone. If it is removed after the ridge has healed, a semicircular incision is made over the area to be removed, the anterior and posterior limits of the incision being 5 to 10 mm. past the undercut area and to the buccal, the zenith of the incision being on the crest of the ridge slightly to the lingual.

3. *Excessively Large Tuberosities.*—These tuberosities present undercuts to the buccal or palatal surfaces, or both. The bulbous portion may be soft tissue, bone, or both. If only soft tissue produces the undercuts, a wedge-shaped section of tissue with the bone posteriorly is removed, and the edges are drawn together with silk ligature to reduce them.

When there is a bony prominence on the buccal surface, a flap is turned. The incision is made from the posterior buccal surface of the tuberosity, running anteriorly and over the ridge to the palatal surface of the crest, and then over to the undercut area sufficiently to allow clear exposure of this region. The flap is raised and held buccally with a periosteotome, while the bone is removed with a chisel, side-cutting rongeurs, and a file. The relationship of the arches in centric should be noted to determine removal of bone on top of the ridge. The flap is trimmed to conform with the lingual edge of tissue, then sutured. The flap is turned more readily when developed on the palatal surface rather than on the crest of the ridge. This procedure brings the crest into better view. If the amount of bone removal is estimated before making the incision so that the amount of tissue on the flap to be removed can be estimated, a closer adaptation for suturing can be made by making two similar incisions in the beginning, making the space between them the width of the tissue to be removed. It is easier and more accurate to incise the tissue while it is held firmly in place by its fibrous attachments to the bone than when the flap is loose and unsupported.

4. *Torus Mandibularis*.—This condition occurs, as either an exostosis or natural bone formation, as a bony prominence located lingually to the mandibular canine and premolar regions, creating severe undercuts on the ridge, and preventing proper denture construction or adaptation of lingual bars for partial dentures. These nodules may be exposed by three adjoining incisions. The first is made along the crest of the ridge extending slightly past the antero-posterior extent of the nodule. The second and third lingual incisions are made one from each end of the first, diverging inferiorly through the mucoperiosteum, and stopping just short of the sublingual tissue attachment to the dental ridge. If there are teeth in this area, the first incision may be supplemented by a simple retraction of the gum tissue from the lingual surfaces of the teeth. The flap is loosened and deflected lingually, exposing the nodule. The nodule may be notched with burrs in the form of furrows, and chiseled off to a smooth area, or the entire nodule may be removed with the burr. When the base of the nodule adjoining the ridge is not too broad, one forceful and sharp blow on the chisel directed at the point of junction with the ridge will be sufficient, leaving only a roughened area to be filed smoothly. The flap is replaced after necessary trimming and sutured to position.

5. *Prominent Knife-edge Mylohyoid Ridge*.—It is best to remove the prominent mylohyoid ridge at the time of extraction. When a patient presents unable to wear dentures because of this ridge, it must be removed and comfortable dentures must be constructed. With a sharp knife an incision is made along the central part of the crest of the ridge from the angle forward to the premolar or canine region as the amount of trimming indicates. The lingual mucoperiosteum is stripped and deflected toward the tongue, exposing the mylohyoid ridge. Remove the ridge undercut with instruments indicated by its size and density. The mucoperiosteum is closely adapted to the lingual wall of the ridge, trimmed, if necessary, and sutured. Greater care should be exercised when working in this area, being careful not to traumatize the floor of the mouth and sublingual structures. There is a greater possibility of post-operative complications in this area, due to lack of drainage and settling of all oral contents into this region.

6. *Torus Palatinus*.—This condition is the result either of malunion of the palatal processes or simple exostosis. Seldom is an undercut produced, but

the bony nodule must be removed to prevent difficulty in proper denture construction. In correcting this undesirable condition, a midline incision is made over the nodule extending anteriorly and posteriorly well past the area in question. Two diverging incisions are made from the ends of the median incision: the anterior section retaining the blood supply of the nasopalatine artery, and the posterior section that of the anterior palatine artery. The posterior diverging incisions should run toward the posterior palatine foramina, thereby avoiding severance of major blood supply and affording maximum exposure of the operative area when the flaps are retracted. The bony mass or nodule is removed with a No. 10 round surgical burr until the palate is the desired shape. The flaps are adapted to the bone and trimmed closely to the approximate edges and then sutured. This tissue should be pressed firmly into the palate and held under pressure for several minutes. A liquid diet should be followed for a number of days.

B. *Tissue Attachments.*—

1. *Labial and Lingual Frena.*—When a frenum attachment is high on a ridge, it interferes with proper extension of the denture rim. Clipping of the frenum and preventing reattachment are indicated. In simple cases clipping with scissors is satisfactory. Touching the raw edges with iron subsulfate will prevent reunion. In more extensive cases a triangular section of tissue is removed. The upper and lower borders of the frenum are clamped with hemostats and the tissue between them removed. Transverse sutures near the upper and lower borders of the cut will close part of the wound. The patient is instructed to use antiseptic mouthwash frequently.

2. *Muscle and Musclelike Attachments.*—When muscles or strong bands of tissue are attached close to the crest of the ridge, they are cut in the same manner as the frena, suturing being necessary. If the attachment is heavy and extensive, after cutting it may be necessary to construct a baseplate to be worn to prevent reattachment.

3. *Creating Ridges.*—What should and can be done for the patient who presents with little or no alveolar ridge on which to construct a denture? The cheek is retracted and the muscle and tissue attachments along the ridge are incised close to the bone along the entire buccal and labial periphery of the arch. The incision creates a new cul-de-sac and a new alveolar ridge, and can be retained by wearing a previously made retainer plate.

C. *Flabby, Soft-Tissue Ridge.*—This excess soft tissue may be due to a neoplastic growth, to hypertrophy resulting from trauma, or to failure in properly trimming and suturing tissue flaps after extraction of teeth. Superficial incision of the overhanging tissue is valueless, because a mass of scar tissue will form that is practically as harmful as the loose mass removed. Make an incision so that a triangular section of tissue may be removed; then place the buccal and lingual edges of tissue in apposition and suture.

POSTOPERATIVE TREATMENT

Fortunately, in most of the corrective procedures presented, there are few postoperative complications requiring treatment. Discoloration of the tissue requires only an explanation to the patient. After trimming of the mylohyoid ridge, there may be some swelling, suppuration, pain, and temperature. Application of cold packs externally and packing open the wound internally is the

indicated treatment, and the condition usually subsides in three or four days. Rest and proper diet are adjuncts to proper healing. Most postoperative treatments will consist of irrigation and spraying of the suture lines.

CONCLUSION

We have presented several types of surgical corrective measures designed to aid the prosthodontist in the construction of more successful dentures. If all of dentistry will accept the responsibility, advantages, principles, and operative procedures, the present era of dentistry will be remembered by patients and professional men for its efforts in clinical and laboratory advancements, improving abnormal and undesirable conditions of the oral cavity for restorative work, and improvements in technique to accomplish the desired results.

FRACTURES: OPEN REDUCTION VS. SKELETAL FIXATION

JULIOUS R. BOURGOYNE, B.S., D.D.S.*

DEFINITIONS

OPEN reduction is the act of laying open the soft tissues by incision so that the bone fragments at their fracture line may be dealt with and placed in their normal position while in full view of the operator.

Skeletal fixation is the act of immobilizing the bone fragments by screws or pins placed into them from without, these fragments being immobilized by bars held rigid by common attachments.

SYNONYMS

The open reduction method may also be called interosseous wiring, direct wiring, or bone plating. The skeletal fixation method may further be termed extraoral fixation or pin fixation.

GENERAL DISCUSSION

In the clinical study of fractures, one ventures upon many experiences which may serve to alter opinions and ideas on methods of fracture fixation. Observation and statistics give great evidence that the most popular method of our present day is intermaxillary wiring. Honest admission must give this method due credit for more closely approaching the ideal when seeking a universal procedure. There are times when this method is not practical, however, and is definitely seen to be of little value after the case at hand is carefully studied. Necessity then calls for a device that will overcome the shortcomings of intermaxillary wiring.

Some of the conditions that render intermaxillary wiring impossible, or of little value, are as follows:

The Edentulous Arch.—When either or both arches are edentulous, the intermaxillary wiring method is rendered helpless, due to the lack of anchorage

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for the wires. I have, in some few cases, wired an intraoral splint through the bone (Figs. 1 and 2). Though this was found to be of some value, it was very impractical and often brought about slight infection, causing drainage of pus into the mouth. If the wires were tight enough to hold position, they caused enough tension on the raw surfaces to aggravate them and stimulate irritation. If the tension were released, then there was movement at the fracture area.

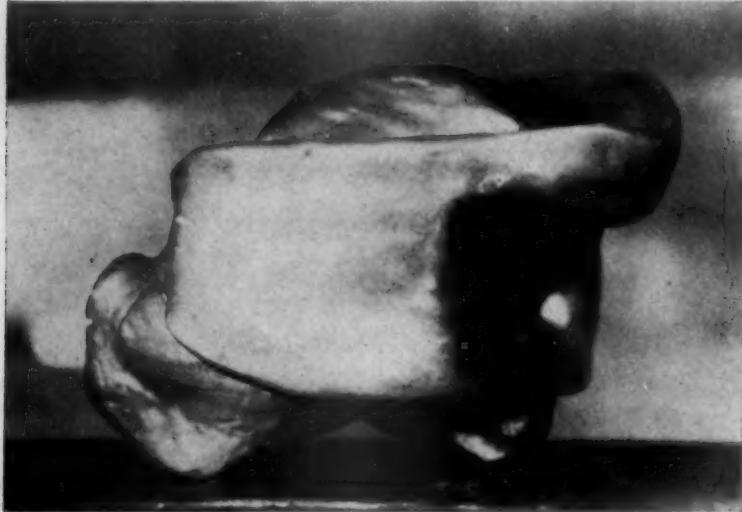


Fig. 1.—Type of splint used intraorally.



Fig. 2.—Type of bandage utilized when the intraoral splint is not wired in position. The bands connecting the head cap with the chin cup are made of rubber dam. The chin cup is made of modeling compound with two tongue depressors passing through it.

Fracture Line Distal to Areas of Dentition.—The angle of the mandible immediately distal to or through the socket of the third molar tooth is a region which is frequently found entertaining lines of fracture (Fig. 3). When this incident occurs and the fracture line slants downward, from anterior to posterior, there is little value in wiring the teeth (Fig. 4). On contraction of the masseter and temporal muscles the posterior fragment or the ramus is pulled

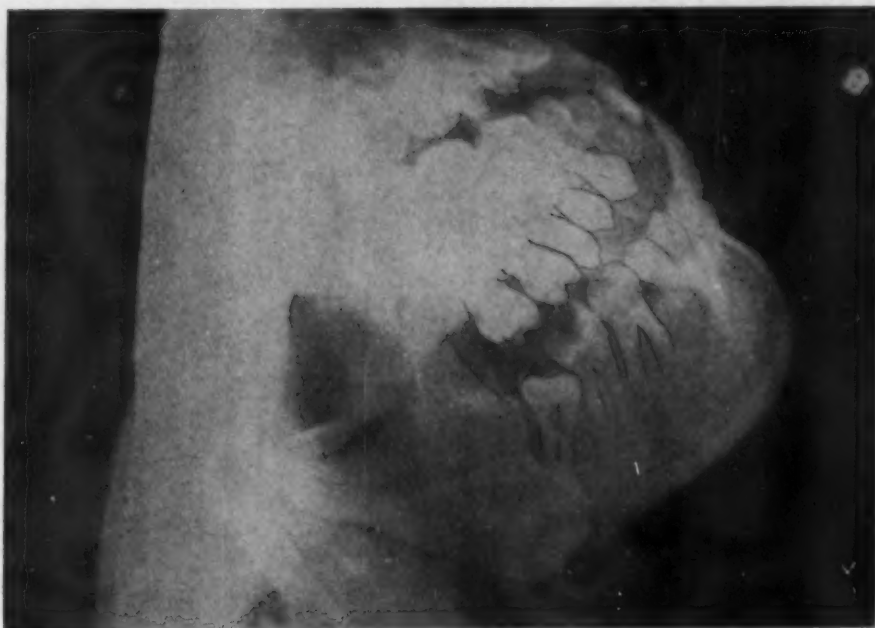


Fig. 3.—A common fracture site through the third molar tooth.



Fig. 4.—The type of fracture in which intermaxillary wiring is of little use.

upward, thus giving the effect on the joint of a closed bite. At the same time, very often the pterygoid muscles pull these fragments slightly toward the median line (Fig. 5). This condition serves to further disrupt the balance of the joint. On healing, if this occurs, though the teeth may enjoy their previous "normal" occlusion, there is what might be called a distorted joint, which very often terminates in pain radiating from the ears due to the pressure of the head of the condyle on the tympanic plate of bone.

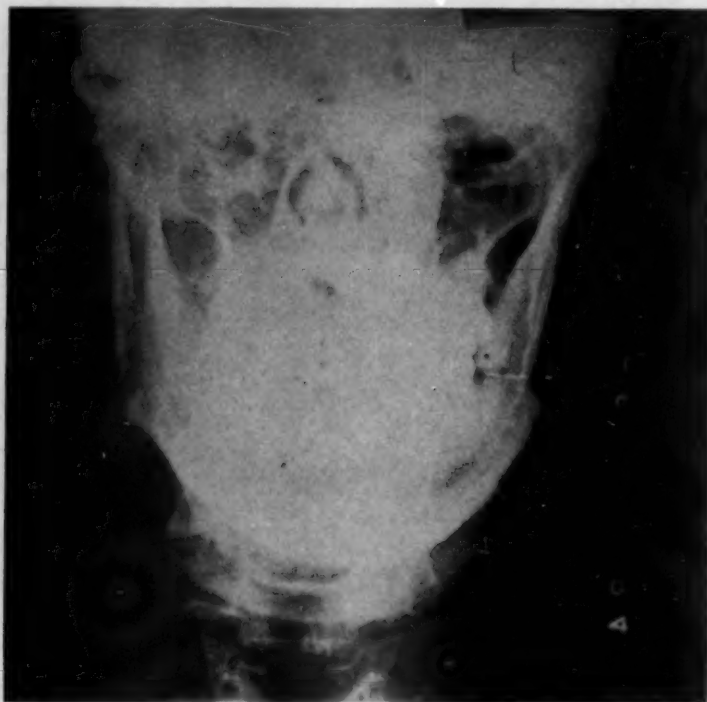


Fig. 5.—This ramus is pulled toward the median line by the pterygoid muscles.

Patients Liable to Nausea.—Transportation is often necessary by air or water immediately after fixation is done. Due to the fact that many people become extremely nauseated with the rocking of a plane or boat, some type of appliance should be used that will allow the patient to open the mouth when necessary, in order to expel regurgitated material from the stomach. During regurgitation this material is poured into the mouth from the stomach faster than it can exit between or around the teeth. Therefore, it is necessary that the jaws enjoy their freedom. The advocacy is made by some operators to use the intermaxillary elastic band method (Fig. 6), then simply remove or cut the bands when nausea occurs. This idea is plausible; however, sometimes nausea occurs so suddenly and in such volumes that the patient's life is endangered by aspirated vomitus before the bands can be loosened.

There have been some few cases under my observation where the teeth were wired by the intermaxillary method under general anesthesia, due to the great amount of pain involved, and had to be immediately cut free on return of the reflexes, because of the occurrence of nausea. The three above-mentioned conditions are those requiring fixations other than intermaxillary wiring. However, there are several conditions that prevent this method from being that of an ideal nature. Some of these conditions are listed as follows:

1. Inability to keep the mouth and teeth normally clean.
2. The muscles of mastication are deprived of their average exercises, thus

causing the movements of the mandible to be limited for some time after the wires are removed.

3. The diet must be severely changed, limiting the intake to liquids or semiliquid material.

4. The wires, in the majority of cases, do some damage to the gingival attachment or to the teeth themselves.

5. Good speech is hindered.

6. Normal expressions are limited to some degree.

In selecting a method for use in place of intermaxillary wiring, one should weigh the facts and history of the case carefully before a final choice is made. It is the opinion of this author that one of the following methods, according to the facts of the case, should be that of choice: (1) Open reduction. (2) Extraoral fixation.

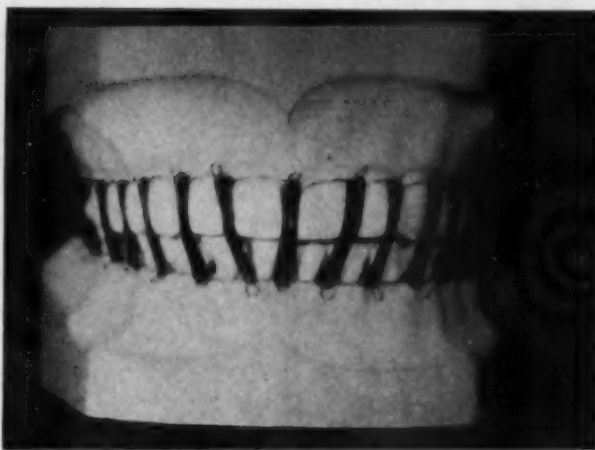


Fig. 6.—The elastic band technique.

The choice of one of these methods, since extraoral fixation was devised, has been the subject of heated and lengthy arguments. Those in favor of open reduction state that there is no need for the patient to wear the unsightly appliances which are offered by the skeletal fixation method. They also state that pins or screws opening into raw surfaces, for the period of time necessary for union of bone, cause infectious materials to be thrown off into the blood stream, thus causing various disturbances. On the other hand, those opposing open reduction argue that this method leaves a scar on the face, and when the appliance is left in position permanently it is definitely a foreign body which may act as an aggravator or irritant at any future date.

We are shown by the experiences of many that neither argument can be discredited. We simply must weigh the two, extract and use the best points from each, or choose the method designated for each particular case.

It is the opinion of this writer that open reduction should be used in many cases; however, first, the operator must understand how to choose each case where open reduction can be used successfully, and why it sometimes fails. On mandibular fractures, for instance, it cannot always be used with as much success as skeletal fixation, due to the fact that these fracture lines often open into the mouth. Cases of success are rare when any foreign body regardless of its make-up, including the foreign bone graft, crosses a fracture line which communicates with the mouth.

In order to understand the argument more fully, the following paragraphs discuss the advantages and disadvantages of the two methods.



Fig. 7.—Open reduction method using floral wire for fixation. In this case the wire passes inferosuperiorly through the bone instead of buccolingually.



Fig. 8.—Open reduction fixation using wire at the angle of the mandible.

OPEN REDUCTION

Before the use of the extraoral splint became popular, fracture fixation using the open reduction methods, by direct wiring and plating, was the principal procedure used on edentulous cases. Most of our plastic surgeons today consider this the method of choice, thus eliminating all visible contraptions. However, it has a great disadvantage in that the wire or plate must be placed across or through the fracture line; if the fracture is compounded into the mouth

Fig. 9.



Fig. 10.

Fig. 9.—Fracture at the mental foramen region of an edentulous mandible.
Fig. 10.—Open reduction fixation of the fracture shown in Fig. 9 by wiring.

the case will seldom, if ever, survive without infection. Also, there have been numbers of cases which gave trouble long after the case was apparently well; this trouble, of course, developed from irritation given by the foreign body imbedded in the tissue. These instances of delayed infection are not common but are sometimes found.

The kind of metal wire, plate, or screws, seems to make little difference if the wound is not a compounded one, and if the material is aseptically buried in the tissues. Several different metals have been used by this author in open reductions, including the regular floral wire taken from the stems of flowers from the florist (Fig. 7). All of these lay dormant in the tissues when they were not first externally contaminated. However, when using metal in the form of screws or pins, which extend into the tissues with a portion remaining without, whether it be through mucous membrane or skin on the outside of the face, the vitallium screw definitely shows a great advantage as the tapered vitallium screw seldom causes rarefaction of the bone.

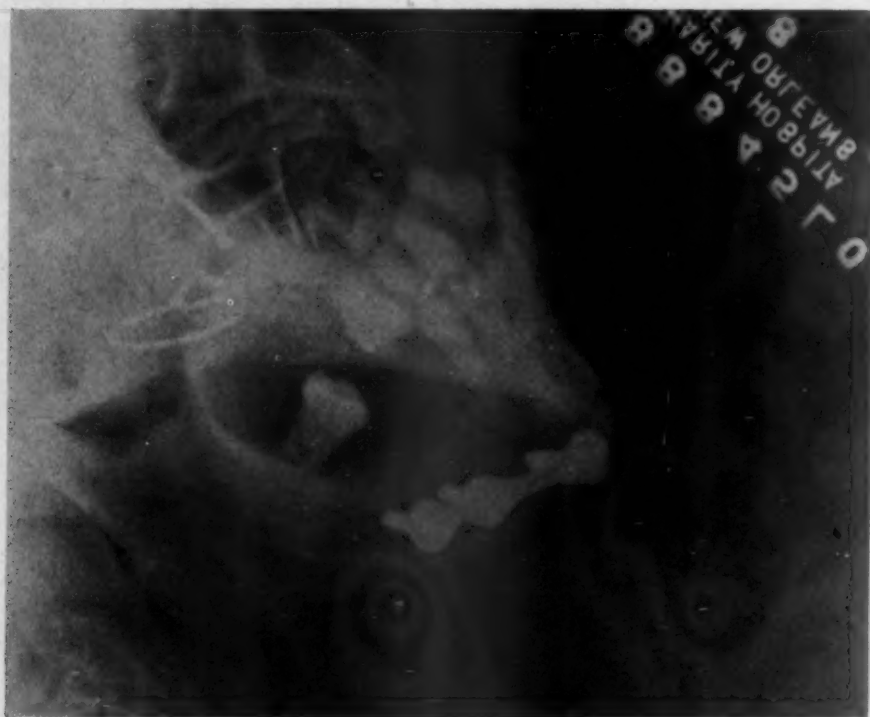


Fig. 11.—Open reduction method using the Lane plate and screws.

The open reduction fixation is fairly stable when the wire or plate is correctly placed. The position and direction of the break will, in some cases, determine a definite position of the wire or plate.

Direct wiring or plating of the bone fragments is sometimes done intraorally, but this is obviously an inferior procedure. The wire or plate, in practically every case, must later be removed, and while it is in place pus is constantly being thrown off into the blood stream and mouth, which is a distasteful thought, to say nothing of its being very unhealthy for the patient. Attempts to bury foreign bodies in the tissues from an intraoral approach will, in practically all cases, result in failure.

The disposition of the fracture many times has much to do with the appearance of the postoperative scar on the face left by the incision. A vertical, clean, and simple fracture seldom ever requires a large incision.

Both open reduction and skeletal fixation should be performed under strict aseptic conditions. In either case, entering the mandibular canal will make little or no difference in the prognosis of the condition.

Open reduction has an advantage in the fact that the bone fragments are actually seen and can, therefore by sight, be placed in an accurate position.

Fig. 12.

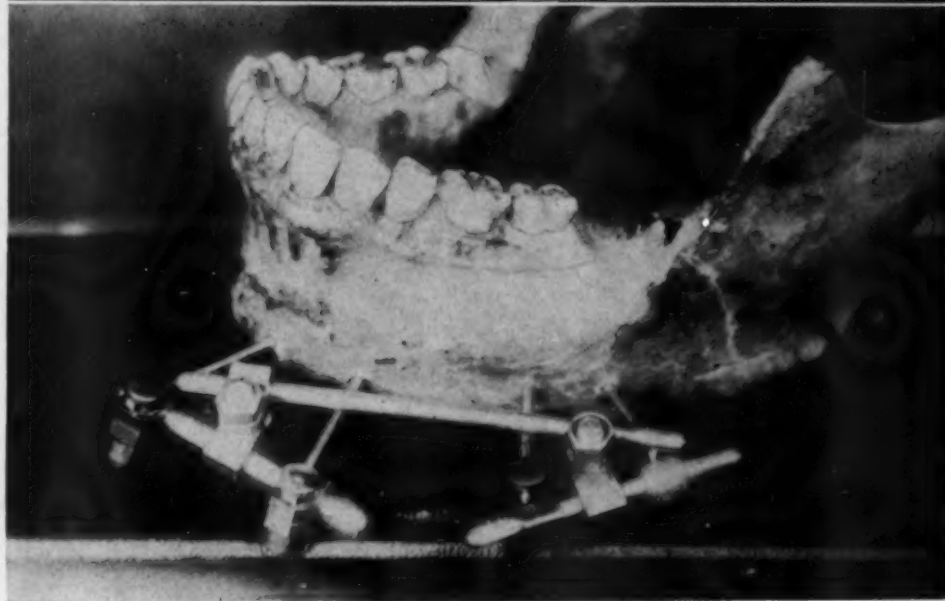
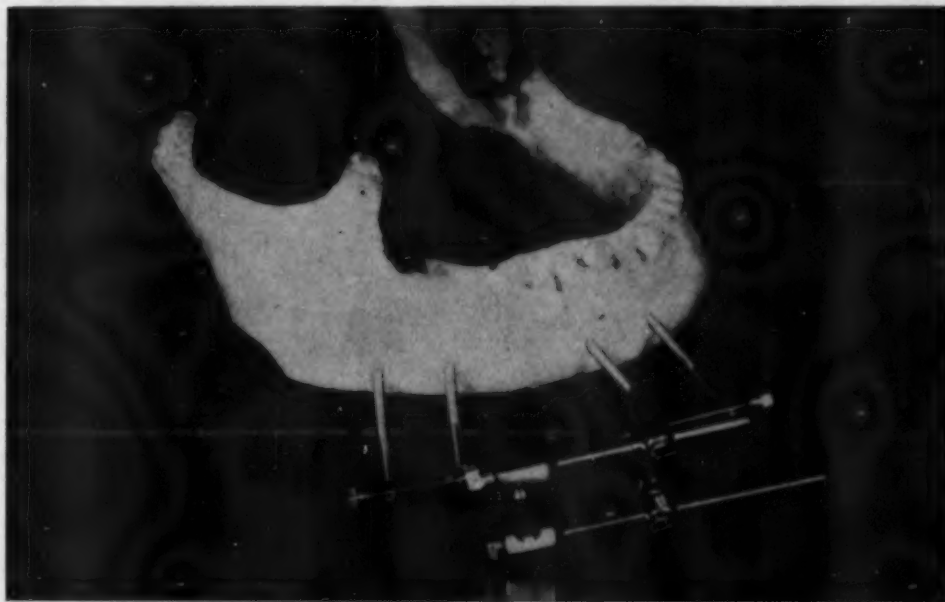


Fig. 13.

Fig. 12.—The Haynes Griffin appliance.

Fig. 13.—The Roger Anderson appliance.

CASE 1.—Mr. M., aged 38 years, entered the hospital for treatment of a bilateral fracture of the mandible, having been sent from a smaller hospital approximately 150 miles away.

On examination it was found that the patient entertained a fracture in the area of the right angle and another in the area of the left mental foramen. The fractures were of two weeks' duration. Fixation using pins in the fracture line was attempted previously at the other hospital, but this information, unfortunately, was not obtained in the history.

That operation, of course, compounded the fracture. An initial dose of 45 grains of sulfa-thiazole was given to be followed by 15 grains three times daily.

There appeared to be no infection whatsoever. The operation was carried out, using

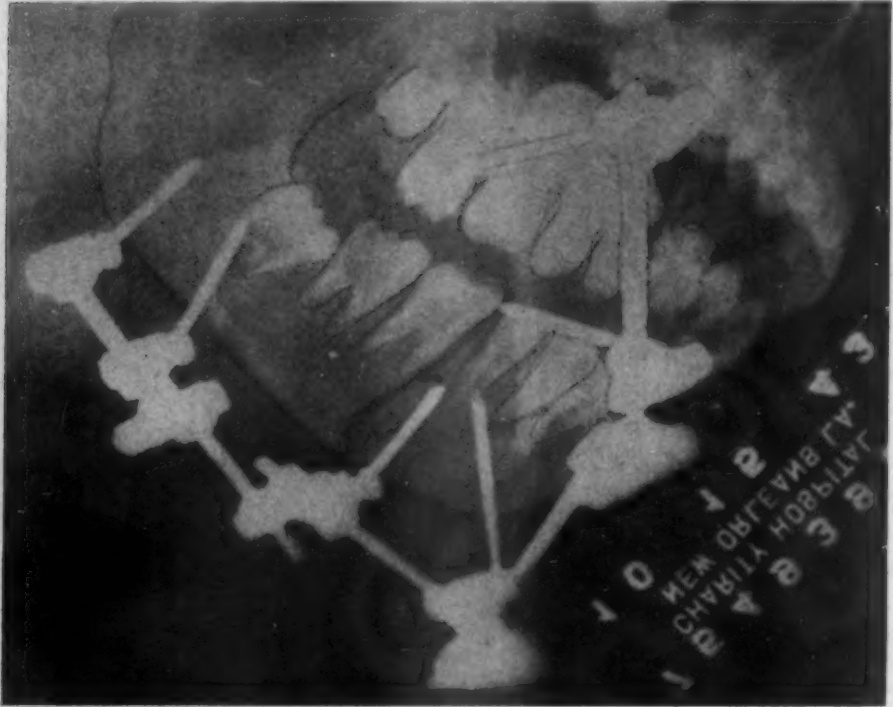


Fig. 14.—Lateral radiographic view of the Roger Anderson appliance in position.



Fig. 15.—Anteroposterior view of the Roger Anderson appliance in position.

silver wires at the right angle and a Lane plate with three screws on the fracture in the left mental foramen region.

On the fourth day, pus was found draining from the area of the right angle of the mandible. On the fifth day, pus was found draining from the area of the left mental foramen. These areas were irrigated with sulfathiazole water and injected with zinc peroxide paste, but the pus continued to flow, and radiographs showed the condition to be worse.

On the twelfth postoperative day, the wires and plate were removed. A one-wrap head bandage was extended under the patient's mandible to hold it in this position to eliminate overactivity for a period of a few days. The draining areas were irrigated three times daily with hydrogen peroxide and sulfathiazole water. After the third day the drainage disappeared.

Three weeks after removal of the plate and wires the fracture lines, which separated the slightly malpositioned fragments, were entered intraorally and the bone ends freshened with the scalpel and bone file. Skeletal fixation was placed on the patient bilaterally and there was uneventful healing.

CASE 2.—Mrs. T. entered the hospital with a bilateral mandibular fracture. The examination revealed the mandibular area slightly swollen bilaterally and a bruised spot over the left mental foramen. Radiographs showed a fracture line at the right angle, and another at the left mental foramen. The history gave evidence that the accident had occurred ten days previously, but there had been no attempt at fixation due to the fact that she had not suspected a fracture. Later there were pains in the ear and the patient could feel crepitation in her lower jaw when attempting mastication.

An initial dose of 45 grains of sulfathiazole was given and an order issued for 15 grains three times daily thereafter for a period of four days. Using silver wire at the right angle and a vitallium plate with three screws in the left mental foramen area, the open reduction procedure was employed. Alternate sutures were removed on the third day and the remaining sutures removed on the fifth day. The wires and plate were left in position and there was an uneventful union of the fragments.

Conclusion.—The above cases are only two among many coming under the observation of this author, which illustrate the fact that there must be a careful choice when the open reduction method is used. Care must always be taken not to enter the mouth from the fracture line in any way. No open reduction should be attempted until all infection at the area of the fracture has completely subsided.

SKELETAL FIXATION

Skeletal fixation is one of the greatest advancements in fracture fixation that we have ever known. Where it can be used, it corrects all of the disadvantages of the intermaxillary wiring method. Good oral hygiene may be obtained, due to free access to all areas while cleaning the mouth. There is no impediment of speech, this being due to normal movement of the jaws, allowing correct position of the tongue, teeth, and lips for pronunciation. The muscles of mastication get practically normal exercise, because of the freedom allowed the mandible when this method is used. There are no irritating factors supported by the teeth and gums. The diet is only slightly changed, and in some cases not necessarily changed at all. The patient is free to expel any regurgitated matter from the mouth which may occur after general anesthesia, or due to air- or seasickness.

The great advantage of skeletal fixation over open reduction is that there is no foreign body contracting or crossing the fracture line to encourage irritation, contamination, and infection. Another advantage over open reduction is that the area of scar in comparison is negligible. It might also be added that, on sufficient union, the appliance may be removed, thus eliminating possible future complications due to the presence of a foreign body.



Fig. 16.—The writer's technique, using the plaster splint.



Fig. 17.



Fig. 18.

Fig. 17.—The plaster splint utilized for stabilizing a multiple comminuted compound fracture of the anterior portion of the mandible.

Fig. 18.—Radiographic view of the plaster splint in position.

No method yet devised can be said to be perfect. We, therefore, must admit the disadvantages of skeletal fixation, the greatest of these being that, in some cases, it leaves small dimpled scars in the skin after healing at the sight of the screw or pin. Careful application of the screw or pin will eliminate some of these incidences. The areas of incision around the pins or screws should constantly be covered with clean gauze to help prevent contamination. Careful and correct application will also eliminate the rarefaction of bone in the area of the appliance. The tapering type screw seems to be of great advantage over other forms.

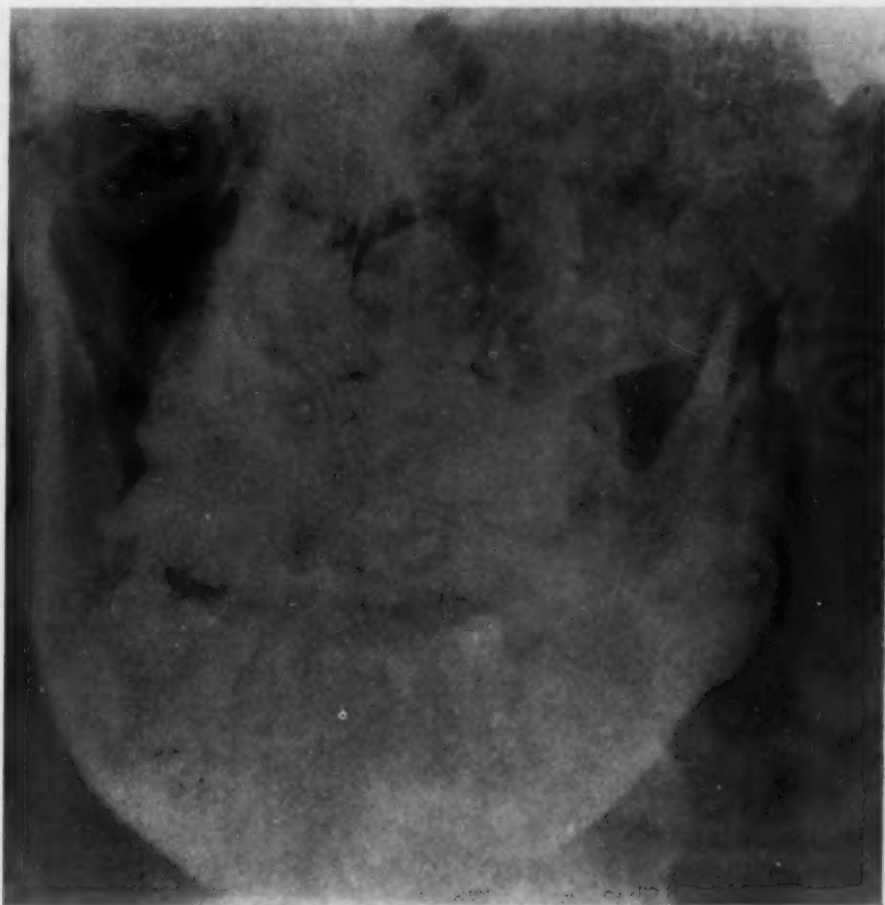


Fig. 19.—Fracture at the neck of the condyle. This type of fracture should not be fixed by open reduction or skeletal fixation.

Skeletal fixation is to a certain extent unsightly. This, too, may be brought to a minimum by careful application and care in choosing the appliance to be used. The author's technique utilizes a splint of plaster (Figs. 16, 17, and 18). Although other techniques similar to this are in use and have been for some time, there is none that offers such simple equipment and assimilation when correctly applied. Its addition to the face is not of such a nature to arouse curiosity among onlookers. For these reasons it is practical for nearly all mandibular fractures.

ANESTHESIA

The open reduction operation and skeletal fixation both may be performed under either local or general anesthesia. However, general anesthesia, using the endotracheal method, is preferable, especially when there has been considerable trauma and where a great deal of soreness is entertained.

CONTRAINDICATION FOR BOTH METHODS

In the opinion of the author, there are several conditions where both methods are contraindicated. One such case is that of a condylar fracture or a break at the neck of the condyle (Fig. 19). In the majority of cases correct position may be attained by manipulation as with a dislocated jaw, and the fixation may be attained by intermaxillary wiring or intraoral splints with a head bandage holding the upper and lower arches in correct position. The danger of open reduction or skeletal fixation, in the above-mentioned case, is that, should infection appear, which sometimes is an occurrence beyond the power of the operator to prevent, the proximity of the temporomandibular joint to the screws is such that ankylosis or involvement of the ears may result.

Another contraindication is where the screws or plate in any way contact the oral fluids. Here, again, a foreign body is found in the tissues under constant surveillance of contamination, irritation, and infectious organisms. Most often even Nature does not let these conditions overlook such lasting and persistent opportunities.

Still another contraindication for the use of these two methods is on thin flat bones of the face. If the bone has any thickness or body to it in the flat bone area, and is not too badly comminuted, and is without contamination by being compounded, then it is permissible to use these methods on the flat bone.

CONCLUSION

Both the open reduction and the skeletal fixation methods play important roles in fracture repair work. Neither can be said to be the best in every case. It is the opinion of this writer, however, that skeletal fixation holds a slight superiority, and, therefore, may be the choice in a great number of cases.

Skeletal fixation has yet great possibilities of rapid advancement, due to improved techniques. At present, as far as fractures of the jaws are concerned, as a universal method it stands second only to intermaxillary wiring.

Advancement is made only by experimentation; therefore, we should not limit our fixation on all cases to one method, but thoroughly search, at all times, for those improvements that are sorely needed.

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SUPERNUMERARY TEETH: ROENTGENOGRAPHIC METHODS OF LOCALIZATION

CASE REPORTS

JAMES M. STRICKLAND, D.D.S.*

INTRODUCTION

ABNORMAL prolific budding of the cells of the formative organs of the teeth, so that teeth numbering in excess of the present evolutionary normal complement are formed, is not too uncommon an occurrence. These extra (supernumerary) teeth, whether examples of atavism¹ or only results of occasional eccentric proliferation of cells of the enamel organ, are in many cases of great concern to the patient and the dentist. Heredity may play its part in the occurrence of these teeth in certain families, but this could not account for the first one in the family history nor for those isolated in a single generation. Stafne² reports on the location of five hundred supernumerary teeth, finding them eight times more frequent in the maxilla than in the mandible, and more common in the incisor and molar areas, as follows:

| | CENTRAL INCISORS | LATERAL INCISORS | CANINES | PREMOLARS | PARAMOLARS | FOURTH MOLARS | TOTAL |
|----------|---------------------|---------------------|---------|-----------|------------|------------------|-------|
| Maxilla | 227 | 19 | 2 | 9 | 58 | 131 | 446 |
| Mandible | 10 | 0 | 1 | 33 | 0 | 10 | 54 |

These teeth have been found in practically every location possible in the dental arches. Their anatomy is most often abnormal, relative to their resembling any particular normal teeth. They are usually smaller than the average tooth in size, and the crowns are conical in shape similar to "peg" lateral incisors. The anatomy and location of these teeth are as unpredictable as the occurrence.

Indications for removal of supernumerary teeth are many and varied. Probably the most urgent indication is in cases where these teeth are factors in the production of malocclusion and must be removed to facilitate and insure successful treatment by the orthodontist (Fig. 1). Of interest to the prosthodontist is the possibility of the supernumerary tooth erupting after artificial dentures are made (Fig. 3). The diagnostician is especially concerned with the possible role of these teeth in cases of neuralgias of the head and neck. The effect that this extra tooth with its eruptive potentiality will exert upon the closely associated normal tooth is the question facing the general practitioner, especially when he anticipates using the normal tooth as a bridge abutment. (Fig. 4). The pathologist is concerned about possible cystic or neoplastic formations resulting from eccentric or otherwise stimulated proliferation of the enamel organs around the crowns of these teeth, either before removal or after incomplete removal of the formative organs of the tooth. The surgeon is interested then in the location and the most advantageous approach for removal of these teeth. The technique of surgical removal depends upon the case at hand and the ability and preference of the operator.

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Roentgenographic Localization.—Roentgenographic localization of the labiolingual and buccolingual position of these teeth is essential to proper surgical removal. One is most embarrassed when, in the attempt to remove an impacted tooth, he opens into the labial alveolar plate and finds no tooth; then he must enter palatally in order to successfully carry out the procedures. The reason given to the patient for this error is most often misleading as to the operator's diagnostic ability and surgical judgment and skill. A very simple roentgenographic technique for locating these impacted teeth is advocated by Clark.³ These supernumerary teeth are usually found by a full mouth x-ray examination or when x-raying possible bridge abutments. The relationship of the extra tooth to some anatomic landmark on this roentgenograph is noted; for instance, the proximity of its root or crown apex to the apex or periodontal membrane of a normal tooth or foramina, suture lines, or antra. Another dental roentgenograph is then made with the rays passing through the area at a different angle to the teeth and the film is directed so that the rays approach the teeth either from a mesial or distal point relative to the approach used in

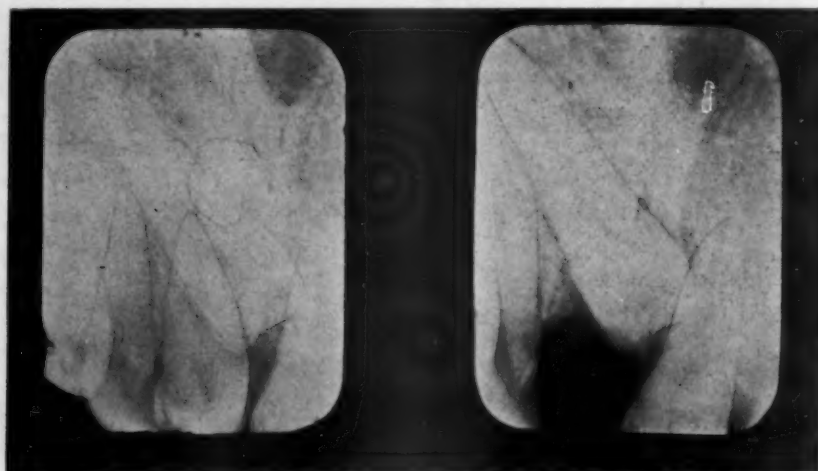


Fig. 1.

Fig. 2.

Fig. 1.—Two supernumerary teeth, central incisors, one erupted so that it caused impaction of the normal central incisor, and the other located superiorly to the latter.

Fig. 2.—Postoperative roentgenograph one month after removal of the two supernumerary teeth.

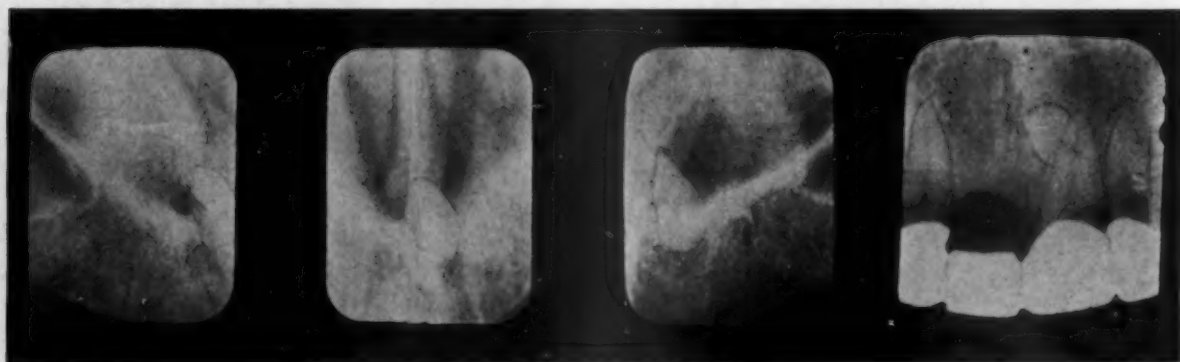


Fig. 3.

Fig. 4.

Fig. 3.—Maxillary anterior views from a routine full-mouth roentgenographic examination revealing impacted supernumerary central incisor. Application of Clark's method of localization (Fig. 5) to these views indicates that when the rays were shifted distally to take the left canine view, the impacted tooth moved distally also, that is, away from the nasal septum that was superimposed upon it in the central incisor view.

Fig. 4.—Supernumerary tooth located near the apex of the maxillary central incisor bridge abutment.

the first exposure of this area. If, for example, in x-raying maxillary central incisors the rays approach these teeth perpendicular to their labial surfaces and indicate on the film a supernumerary tooth between the roots of the central incisors, we have only to move the x-ray tube distally, right or left, so that the rays approach these teeth at an angle acute to one tooth and obtuse to the other. The central rays should enter now through a central or lateral incisor instead of the midline as in the previous exposure. This shifting of the path of the rays through the teeth produces on the film a different relationship of the supernumerary tooth to its surrounding structures. By the proper interpretation of this change we can locate the labiolingual position of the supernumerary tooth relative to the landmarks chosen on the first film.

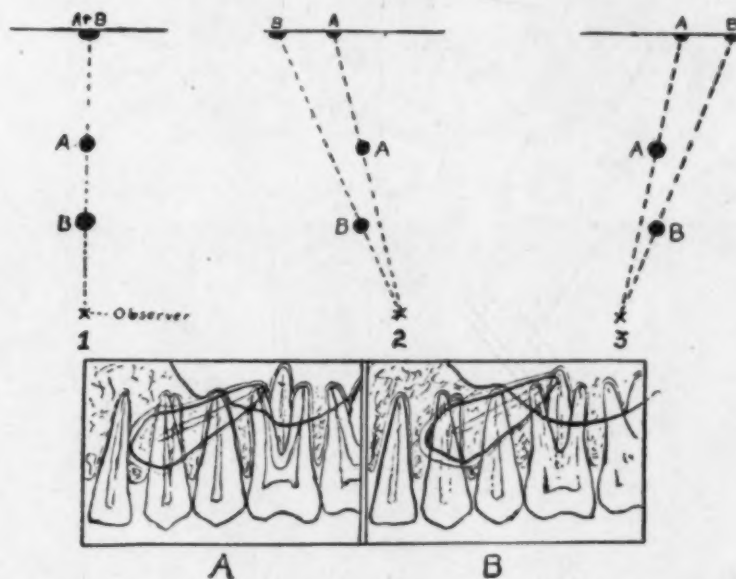


Fig. 5.—Clark's method of localization of impacted teeth and foreign bodies. 1, Two objects in a straight line with the observer; the more distant one is covered by the nearer. 2, Observer moves to the right; the more distant object is apparently to the right of the nearer one. 3, Observer moves to the left; the nearer object is apparently to the right of the more distant one (Ivy). A, Tip of canine touches the second incisor. B, Tube is moved to the right; the tip of the canine is moved away from the incisor and in the same direction as the tube; the canine is, therefore, the more distant object and lies on the lingual side. (From Ennis: Dental Roentgenology, Lea & Febiger.)

If, in our example above, the central rays were shifted distally to enter the left central incisor and on the resulting film the supernumerary tooth is now in closer proximity to the right central incisor than in the first exposure, we can say that the extra tooth moved on the film mesially, in the opposite direction of the distal movement of the rays; therefore, the supernumerary tooth lies labial to the roots of the central incisors. Had it moved, however, on the second film closer to the left central, it would be located lingual to the roots of the central incisors. This principle of interpretation of the movements is most clearly illustrated by Fig. 5.*

When in a dental roentgenograph a supernumerary tooth appears over the apex of a tooth root, Ciezynski's topographical method can be used as well as, or in conjunction with, Clark's method of localization. Ciezynski's method is illustrated by Fig. 6.*

Perhaps a more foolproof method of localization, where utilizable and convenient, is that of a lateral view giving a direct labiolingual relationship of the teeth (Fig. 7). Regular occlusal film packets are of sufficient size to use for

*Figs. 5 and 6 reproduced through the courtesy of Dr. LeRoy Ennis and Lea & Febiger, from Ennis: Dental Roentgenology.

this purpose. Difficulty in securing adequate detail in these views is the only obstacle encountered. Care must be exercised in directing the central rays parallel to a frontal plane bisecting the central incisors labiolingually and perpendicular to the film and sagittal plane. This view is utilizable only when the impacted teeth are located in the anterior segment of an arch. With super-speed occlusal film at a tube-film distance of 20 inches, 10 to 15 seconds exposure will usually give sufficient detail.

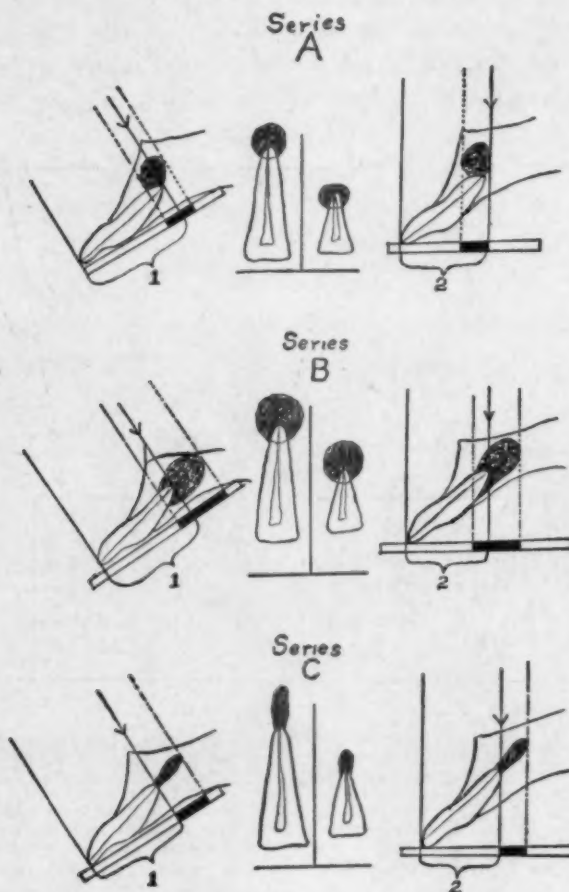


Fig. 6.—Cieszynski's topographical method of localization of periapical lesions and impacted bodies. Two views are necessary, one to show the normal tooth length, and one foreshortening the area. *Series A*, In the diagram showing the tooth of normal length (1), the shadow of the bone cavity appears above the apex of the tooth, while in the foreshortened diagram (2), it appears below the apex of the tooth. From the application of the theories governing shadow projection, it is discovered, therefore, that the diseased area is situated near the labial plate. *Series B*, The shadow appears both below and above the apex of the tooth in both diagrams, and, therefore, the cavity lies near the palate. *Series C*, In these diagrams, the cavity is apparently above the apex and in the direction of the long axis of the tooth. In diagram 1 the shadow of the cavity appears longer and narrower, while in diagram 2 it seems globular and diminished. In neither diagram does the cavity appear to be superimposed over any portion of the root. The area in question, therefore, lies directly apical to the root. (From Ennis: Dental Roentgenology, Lea & Febiger.)

CASE REPORTS

CASE 1.—T. S., a 16-year-old boy, presented with a malformed and malposed maxillary right central incisor, asking for extraction and replacement of this "ugly tooth." The roentgenograph, Fig. 1, was made for routine diagnostic information. It was found that the "ugly tooth" was an erupted supernumerary tooth, the normal central incisor being impacted apically to it, with a second supernumerary tooth located superiorly to this normal impacted tooth. The question then arose as to the possible removal of the impacted teeth and restoration of the erupted supernumerary tooth, or removal of the supernumerary teeth allowing the normal central incisor to erupt either naturally or with orthodontic stimulation.

*Figs. 5 and 6 reproduced through the courtesy of Dr. Le Roy Ennis and Lea & Febiger, from Ennis: Dental Roentgenology.



Fig. 7.—Lateral or sagittal view of supernumerary tooth lying slightly labial to the maxillary right central incisor teeth.

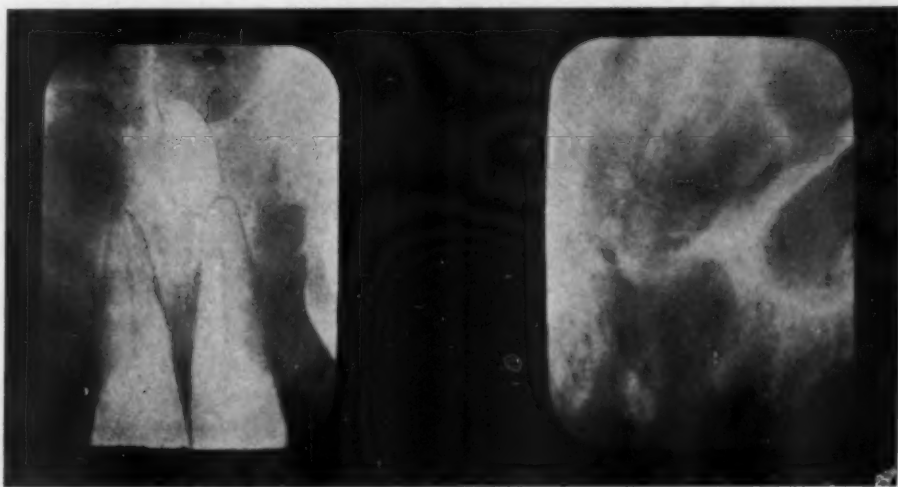


Fig. 8.—Preoperative view of a supernumerary tooth near the apex of the maxillary right central incisor, and postoperative view one month later showing normal healing.

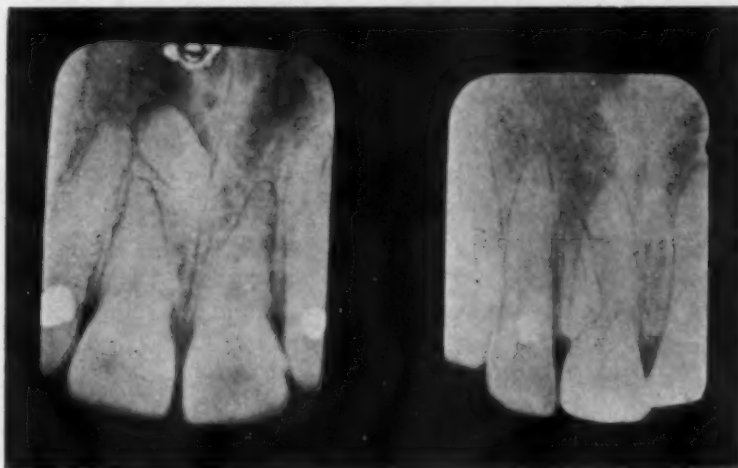


Fig. 9.

Fig. 10.

Fig. 9.—Supernumerary tooth over the apex of the maxillary left central incisor. This is the same case as Fig. 7.

Fig. 10.—Geminate (twin) supernumerary teeth located labial to the maxillary left central incisor.

The latter treatment was instituted. A labial mucoperiosteal flap was elevated from canine to canine. During the surgical operation the roentgenographic interpretation (tentative diagnosis) was substantiated in that the impacted teeth were found in the labiolingual position indicated by the roentgenographs with the apex of the erupted supernumerary tooth too close to the crown of the impacted normal central incisor to permit removal of the latter without serious damage to the apex of the former. Surprising, however, was the finding of the lingual surface of the crown of the normal central incisor facing to the labial. The impacted supernumerary tooth was found deep in the base of the nasal spine as suspected. The operation was followed by no unusual postoperative manifestations. A postoperative roentgenographic view made a month after the operation, Fig. 2, indicates normal healing. The patient became a victim of pneumonia seven months after the operation and died before the eruptive progress of the normal central incisor could be adequately observed.

CASE 2.—J. B. T., a 46-year-old man, presented for full-mouth extraction. Routine pre-extraction roentgenographs revealed the maxillary supernumerary tooth in the anterior segment. This tooth was removed through the alveolar socket of the left central incisor. Postoperative roentgenograph one month after removal of both supernumerary tooth and cystic area apical to the right lateral incisor indicates normal reparative healing. (Fig. 8.)

CASE 3.—R. W., a 35-year-old woman, presented with maxillary full denture of six years' service, and ten mandibular teeth beyond treatment for peridontoclasia. Routine roentgenographic examination revealed an impacted supernumerary tooth in the maxillary anterior segment. (Fig. 3.) This tooth was observed at the time of removal of the maxillary teeth and construction of the denture. The patient was told that the tooth should not be removed, that it never would give her any trouble. The writer does not agree with that opinion. The tooth was removed through a palatal opening. The patient remarked that from what she could remember of the first x-ray view, "the tooth had moved higher." Gentle curettage for the removal of the enamel organ of the supernumerary tooth revealed that the apex of the crown had already protruded into the nasal cavity, there being no separation between them except the nasal mucous membrane which ruptured upon removal of the enamel organ. The flap was sutured and no postoperative complications noted other than drainage into the nasal cavity for the first six to eight hours.

CONCLUSION

Supernumerary teeth are definite sources of abnormalities such as malocclusion and impacted normal teeth. They are potential sources of neuralgias, blindness, deafness, muscle twitching, cysts, tumors, and destructive forces for restored normal teeth, and should not be excluded from diagnostic and prognostic data. The writer recommends removal of all such teeth as a preventive measure for any of the many ills that may result from their presence. Roentgenographic localization is essential to proper surgical judgment and operation, as well as gratifying in its accomplishment.

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Editorial

Mamma Lay That Pistol Down

THE maternal protection of the American College of Dentists is extremely touching. We admit that since the publication of "Status of Dental Journalism in the United States" by the commission appointed by the American College of Dentists, a great deal has been accomplished in the development of dental journalism by the profession and for the profession. The committee apparently takes the view that no journal is to be approved by the profession unless it is published by a dental society. Have we become so regimented that nothing can be considered right unless it is owned and governed by a dental organization? Is there not something to be said for independent journalism in a profession? The medical profession has many independent journals, such as, *Surgery, Annals of Surgery, Journal of Neurophysiology, Medicine*, and *Journal of Laboratory and Clinical Medicine*, all edited by well-known men in the medical profession, and medical men consider it an honor to have a paper accepted and published in these periodicals. Why should not the dental profession have independent journals, particularly if they have a national character? Too many local magazines have been formed during the last decade; every state society, and even fraternity and college publications have started to print scientific papers. Is this necessary to justify the increase in the dues to their members, or is it done to make the journal more important so as to get a greater income from advertisements? I have before me the most recent issue of one of the oldest and best state society journals; it contains 38 pages of text and society announcements, and 23 pages of advertising material of various types, a proportion of sixty out of one hundred, while the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY in the April number contained 152 pages of text and 17 pages of advertisements, a proportion of eleven out of one hundred.

The contents of many of these journals consist mainly of society news and announcements, but a paper and some abstracts are generally included. Papers published in this manner are a total loss, since no investigator or author can review them all, and libraries do not collect and bind them unless they have a particular interest in one of them.

The report of the American College of Dentists makes much of the "control" of a journal. I wonder how much investigation has been undertaken to find out how an independent journal is controlled. As an editor of our JOURNAL, which they have attacked, I personally have never had an inquiry of any sort by any of the members of the present investigating committee regarding control or any other matter pertaining to the JOURNAL. A very thorough investigation that!

The committee makes the statement that since the creation of the *Journal of Oral Surgery* a noticeable change has occurred in the number of literary contributions published in the Orthodontic Section of our JOURNAL during the first six months of 1944. "It is singular," they say, "that only sixteen contributions were published in the Orthodontic Section while the Oral Surgery

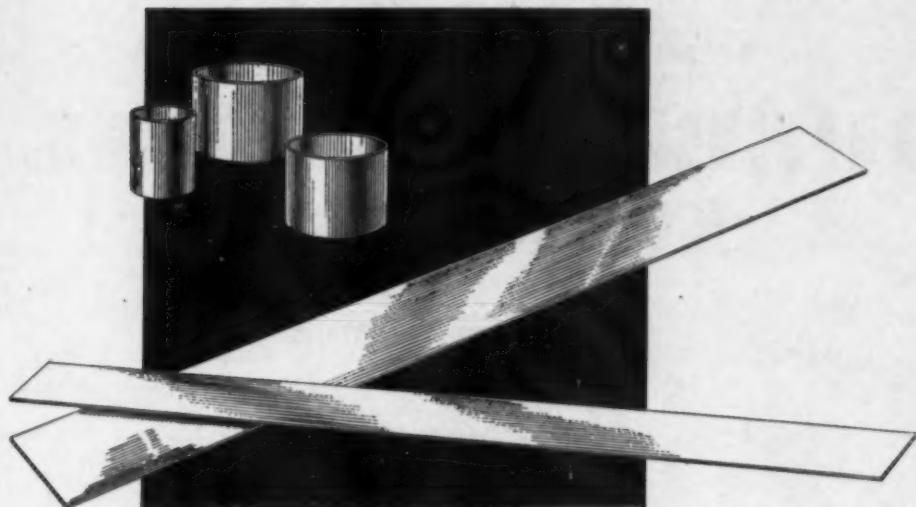
Section contained forty-four." Imagine counting the number of contributions instead of the number of pages, but of course it makes a much better story than the actual facts, which are the following: the Orthodontic Section contained for the entire year of 1944 702 pages of text, while the Oral Surgery Section contained 806 pages. The implication that the return to the owners is being jeopardized through the competition of the *Journal of Oral Surgery*, and, therefore, that "more effort is being made to feature the Oral Surgery Section," I found extremely humorous. I wish the War Production Board would allow us to increase the number of pages still more.

The average dentist is not as gullible as the committee of the College makes him out to be. On the contrary, he recognizes a good honest value. He would not subscribe to any journal because of an advertisement which lists a large number of pages; he wants to see the quality and presentation of the material, and he is willing to pay for what he wants.

Since the committee of the American College of Dentists has raised the question of cost to the subscriber, stating that they feel there is no justification for such a large specialty group as the orthodontists to pay \$6,078 for the JOURNAL per year, let us see how that works out in comparison to the American Dental Association controlled *Journal of Oral Surgery*. The members of the American Society of Oral Surgeons, in 1944, paid \$5.00 for four issues which contained 384 pages of text, while the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY furnished its subscribers in the same year with 1,508 pages in the twelve numbers published for \$8.50. The *Journal of Oral Surgery* would have to sell for \$19.60 if it contained the same amount of material as the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY, figuring the price per page at the rate charged by the American Dental Association. In addition, I am informed that the actual amount paid by the American Association of Orthodontists according to the auditor was not \$6,078, but \$3,515. To the committee this is probably an unimportant error, but it is in keeping with the rest of the report.

We want to assure our readers that the control of all published material is entirely in the hands of the editors and that the editors are trying their best to constantly improve the JOURNAL and furnish the reader with the best material available. Perhaps the committee of the American College of Dentists does not possess enough imagination to visualize that some credit is due the editors for developing a journal, for attracting good, scientific material, and presenting it in a concise, practical manner. They do not like to admit, perhaps, that a well-edited journal may get a reputation which will make it attractive to prospective writers as well as to interested readers. In an independent journal the control of publication rests entirely with the editorial staff, and it is the editorial staff which makes or breaks a publication. An independent journal has no ties, and no politics govern the publication of the material received. The AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY is to be congratulated. It has a board of associate and consulting editors who are recognized leaders in the profession. We are proud of the material that we receive from our distinguished contributors and we know that our subscribers are pleased with the papers the JOURNAL has published. Like the inhabitants of Solomon's house in Francis Bacon's *New Atlantis*, "We maintain a trade not for gold, silver, or jewels, nor for silks nor for spices nor any other commodity of matter, but only for God's finest creature, which is Light."

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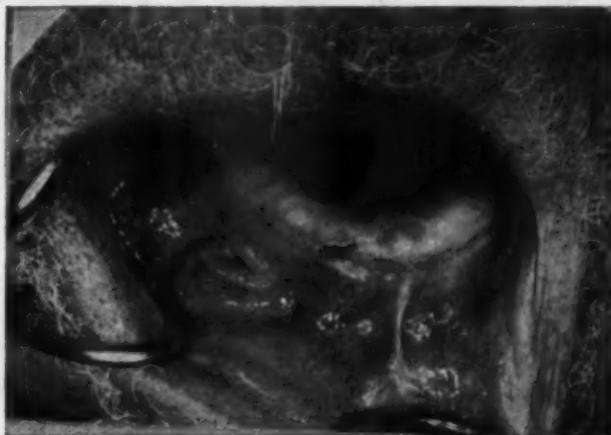
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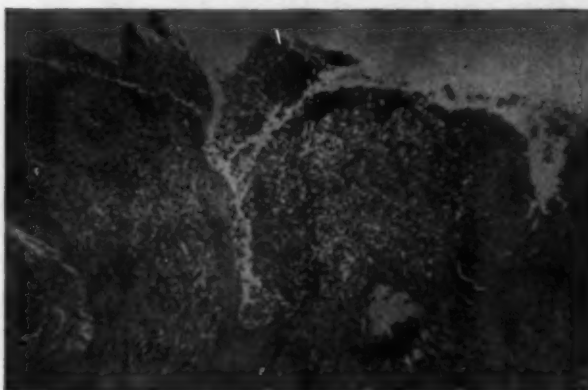


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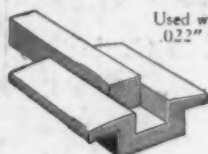
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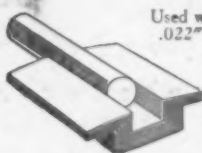
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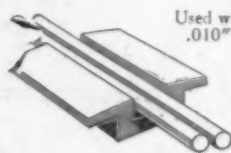
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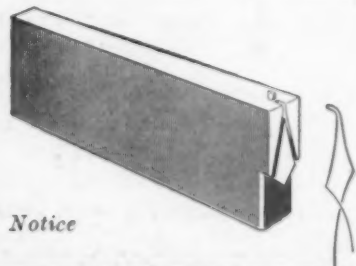
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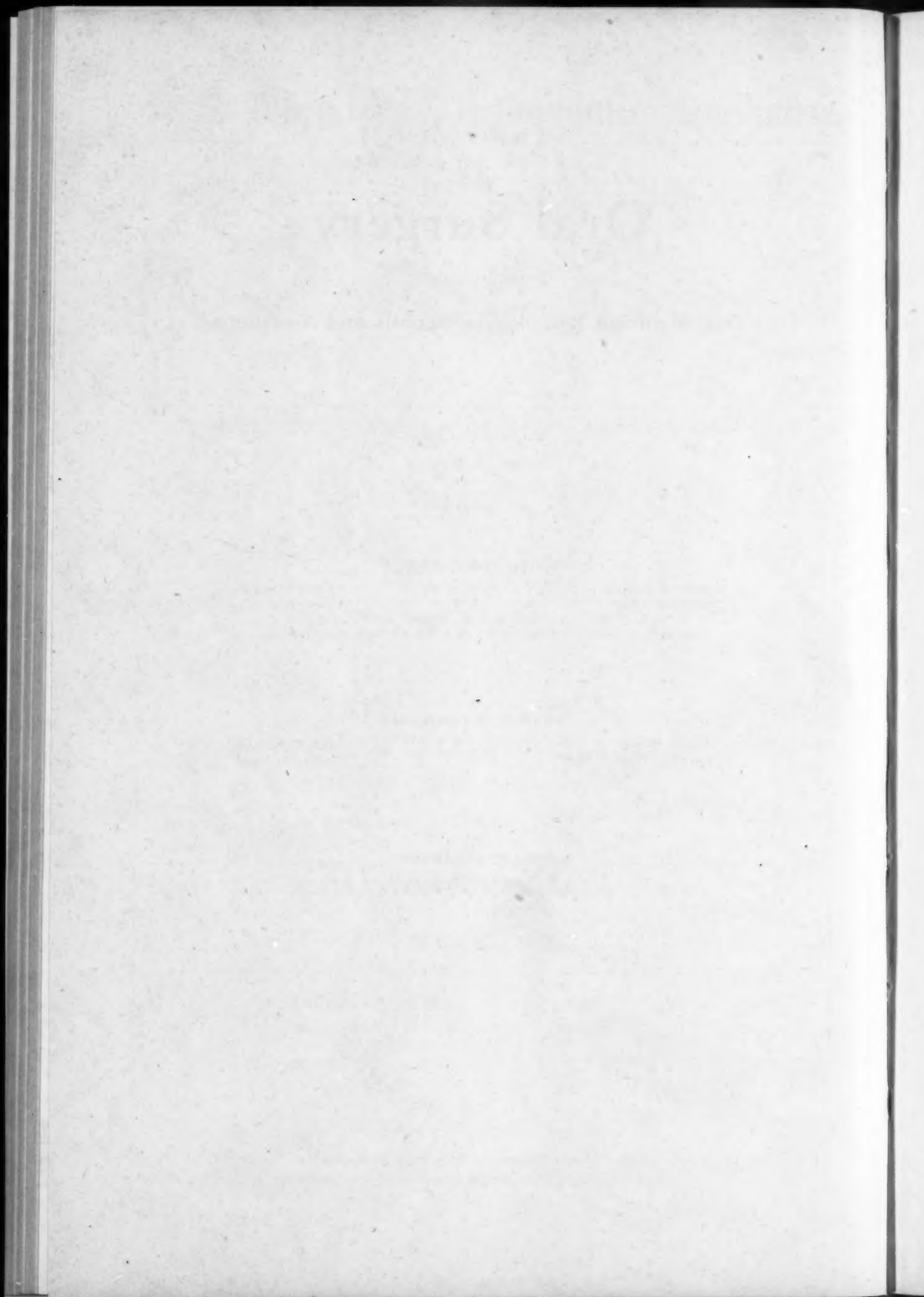
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Ellis Fischel State Cancer Hospital Number

SYMPOSIUM ON NINETEEN CASES OF BENIGN AND MALIGNANT LESIONS OF THE ORAL CAVITY, FROM THE ELLIS FISCHEL STATE CANCER HOSPITAL, COLUMBIA, MISSOURI

Volume II

WILSON N. BURFORD, D.D.S.,* AND LAUREN V. ACKERMAN, M.D.†

INTRODUCTION

THIS second symposium from the Ellis Fischel State Cancer Hospital serves to emphasize the variety of lesions, neoplastic and suggesting neoplasms, which occur in the oral cavity. Each individual neoplasm also varies in its behaviour and stage of development and, consequently, its treatment. About 50 per cent of our cases first consult a dentist, and his knowledge of such lesions, particularly from the standpoint of recognition, becomes of paramount importance, for early diagnosis parallels high percentage curability of oral cavity neoplasms.

I. PLASMA-CELL MYELOMA WITH INVOLVEMENT OF THE GUM OF THE MANDIBLE

Case 21, E. G., EFSCH No. 6617

A 72-year-old white man was first seen in the clinic in July, 1944. A painless swelling in the anterior aspect of the left mandible had appeared three to four months before and had progressively but painlessly increased in size. The local physician referred the patient to an oral surgeon who biopsied the lesion.

Diagnosis.—Transitional-cell carcinoma.

The patient was referred to this hospital. There had also been a mass in the region of the testicle for about five years which for the past few months had increased in size.

Examination revealed a visible deformity of the lateral aspect of the left lower lip. Situated on the mandibular border (beginning approximately 1.5 cm. lateral to the midline and extending distally about 3.5 cm.) was a non-ulcerated, bluish-red, semicystic lesion apparently arising from the mandibular body (Fig. 71). The remainder of the mandible was negative and there were no palpable cervical lymph nodes. The only other positive finding was a firm mass in the region of the spermatic cord, measuring 6 by 4.5 by 5 cm., with enlarged regional inguinal lymph nodes.

Roentgenogram examination of the mandible revealed an irregular and incomplete area of bone destruction in the anterior alveolar two-thirds of the left mandibular body (Fig. 72). A right lateral view of the skull revealed an occasional zone of ill-defined increased translucency in the superior portion of the parietal region and in the ramal plate of the temporal bone (Fig. 73). The

*Oral Surgeon, The Ellis Fischel State Cancer Hospital, Visiting Lecturer in Oral Pathology, St. Louis University Dental School.

†Pathologist of the Ellis Fischel State Cancer Hospital, Assistant Professor of Pathology, Washington University School of Medicine.

ribs and vertebrae were apparently negative. A biopsy specimen was taken from the lesion in the oral cavity.



Fig. 72.—Oblique roentgenogram showing radiolucent area in left mandible.



Fig. 73.—Lateral study of skull showing punched-out areas in calvarium.

Microscopic Description.—Beneath the overlying epithelium a very cellular tumor was present. In many instances the cells had eccentric nuclei with a cart-wheel arrangement of chromatin and pink cytoplasm. A few of these cells were multinucleated. Mitotic figures were moderately frequent and the tumor was well vascularized.

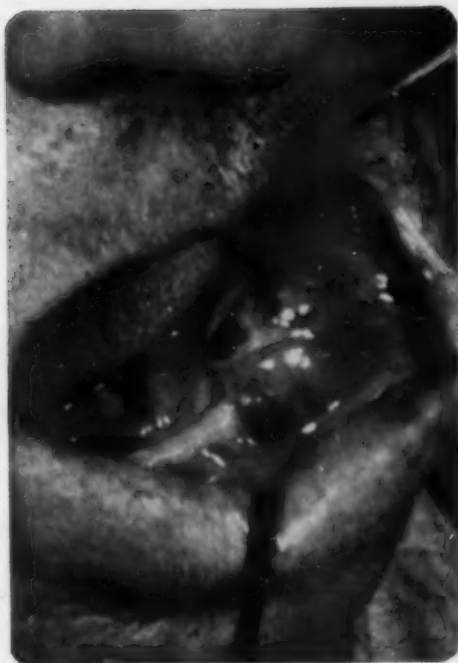
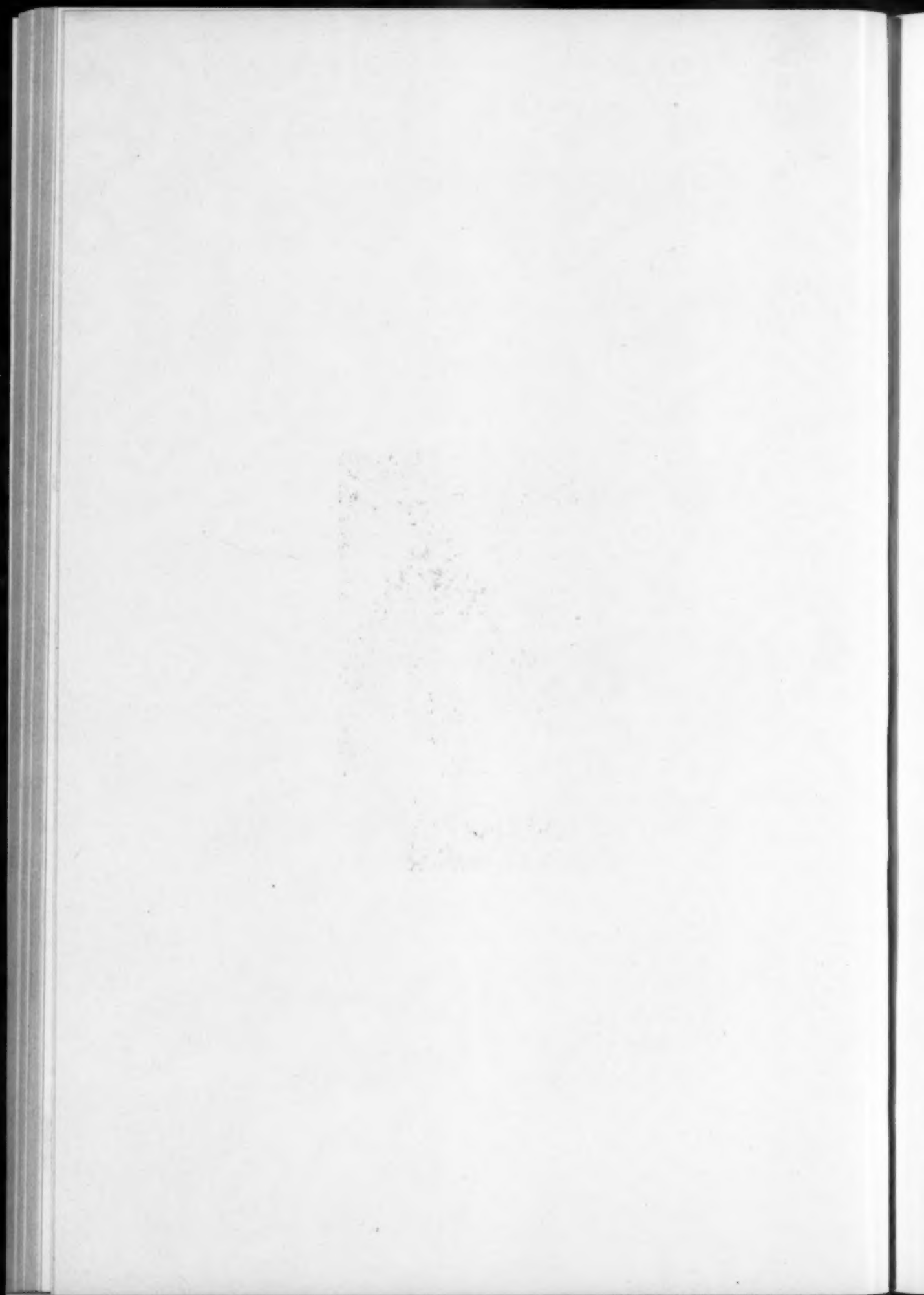


Fig. 71.—Photograph showing nonulcerated, rounded tumefaction on gum of the mandible.



Microscopic Diagnosis.—Oral cavity, alveolar ridge, lower: Plasma-cell myeloma (Fig. 74).

Aspiration biopsy of the right spermatic cord mass was performed on August 2. The microscopic picture was similar to that seen in the oral cavity.

Microscopic Diagnosis.—Plasma-cell myeloma, questionably primary. This diagnosis was further substantiated by the laboratory finding of a serum total protein of 11.5 per cent with 8.6 per cent gram globulin and 2.9 per cent albumin.

It was felt that radiotherapy to the bone lesions at this time would be useless, and the patient was discharged from the hospital on August 8 with no return date for follow-up. If any metastatic bone lesions became painful, palliative radiotherapy would be given to those areas. The patient expired at home in September, 1944.

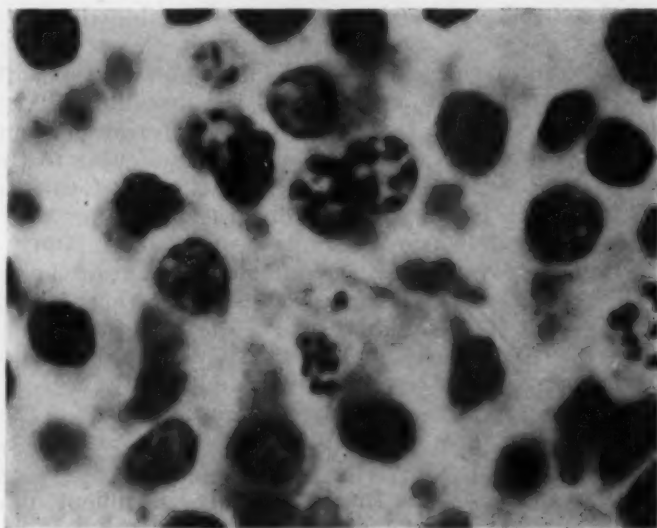


Fig. 74.—Photomicrograph (high-power). Note eccentric nuclei, arrangement of chromatin and multinucleated form.

Comment.—Lesions of the gum of the mandible such as this are distinctly unusual. Biopsy revealed the typical histologic picture of a plasma-cell myeloma. This unexpected finding prompted further laboratory procedures, and x-rays proved that the oral cavity lesion was only part of a generalized process.

The upper respiratory tract is the most common location of extramedullary plasma-cell tumors. Hellwig¹ has reviewed these cases and tabulated 64, of which the majority occurred in the nasal cavity, nasopharynx, and tonsillar region. There were none from the gum of the mandible, but two arose from the floor of the mouth and one from the tongue. Most of these cases were followed for too short a time to be of any value, but a large percentage were obviously benign. Nine tumors of the air passages metastasized to bone. It is impossible to tell in any given instance from the microscopic picture which case is malignant and destined to metastasize.

The case reported here probably arose from the spermatic cord and later disseminated to bone and appeared in the gum of the mandible. The high globulin was to be expected, but the Bence-Jones protein was negative. It should be pointed out that a high serum globulin and positive Bence-Jones proteinuria do not occur until the disease has become generalized. If the lesion in the oral cavity was single and primary, then intensive radiotherapy would have been indicated, for these lesions are very radiosensitive and rarely radiocurable.

II. EWING'S TUMOR OF THE MANDIBLE

Case 22, C. L., EFSCH No. 6289

Possible Primary Ewing's Sarcoma of the Mandible

A 15-year-old white boy entered the hospital in April, 1944, complaining of a gradual increasing swelling in the left lower jaw which had been present for four months. Loosening of the teeth had been apparent before the swelling appeared. A similar swelling later became evident on the right side of the body of the mandible.

On examination, the lower teeth were in full complement but a general loosening of the premolars and molars was noted. On the left body of the mandible there was a smooth and somewhat painful tumefaction measuring 3.5 by 2 by 2 cm. A second smooth tumefaction near the symphysis measured 1 by 1 by 1 cm. On the right, there was a third tumefaction measuring 2 by 1.5 by 1 cm. This appeared to be slightly crepitant on pressure but there was no regional adenopathy.

Roentgenogram examination showed a moderate amount of soft tissue swelling about the right body of the mandible and the mandibular symphysis. Descending anteriorly into the soft tissues from the anterior portion of the right mandibular body were linear spicules of bone perpendicular to the mandibular surface. Periosteal proliferation was noted along the inferior surface of the right mandibular body. In this area, the proliferation was parallel to the parent bone. Several zones of radiolucency, varying from well- to poorly-defined, were noted in the mandibular symphysis in the anterior portion of the right mandibular body (Fig. 75). Bone demineralization and rarefaction were present in the anterior portion of the left mandibular body, the mandibular symphysis, and the anterior one-half to two-thirds of the right mandibular body. Distortion was noted about the margins of several teeth but there was no definite erosion of the root tips. X-ray studies of the chest were negative for evidence of pulmonary metastases. An aspiration biopsy was taken from the tumefaction at the left symphysis.

Microscopic Description.—This tumor presented a difficult diagnostic problem. A Ewing's sarcoma would have been a possibility but it very rarely appears in this location and its cells are very uniform. This tumor presented great variation in its cells. A Wilder silver stain showed a delicate lacy network of reticulum which is rather unusual in a Ewing, although at times a small amount of reticulum may be present. The most likely possibility was an osteogenic sarcoma.

Microscopic Diagnosis.—Bone, mandible: Possible osteogenic sarcoma (Fig. 76).

Radiotherapy was given to two lateral fields directed at both mandibles, each field measuring about 11 by 7 cm. The factors were 200 kv., 15 ma., 60 cm. TSD, filtration 1 mm. copper plus 1 mm. aluminum. The total dosage was 6,000 r. to the two fields over a thirty-seven-day period. At the time of discharge, June 18, the tumor had slightly decreased in size.

On July 26 the mandibular tumefactions had practically disappeared. There was some symmetrical thickening at the outer aspect of the mandible about the level of the first molar, but the area of the symphysis was normal to palpation. Examination of the oral cavity revealed no abnormalities and there were no palpable submaxillary or cervical lymph nodes. X-ray examination of the mandible showed the disappearance of the majority of the osteolytic changes, diminution of the spicules, and some mild calcification of the whole mandible (Fig. 77).

This patient was last seen in April, 1945. The horizontal branches of the mandible were very thick but there was a decrease in the general volume. The teeth were definitely more secure in their sockets and there was no evidence of metastatic disease and no facial asymmetry.

Comment.—In retrospect, it seems rather unlikely that this tumor was an osteogenic sarcoma because of the dramatic response to radiotherapy and absence

of lung metastases. Microscopic examination had not shown any neoplastic osteoid, which is invariably present in osteogenic sarcoma. The presence of reticulum and the variation in cell size, while not usual in a Ewing's sarcoma, do not serve to rule it out. Further follow-up will undoubtedly reveal the correct histologic diagnosis. This case serves to illustrate the desirability of strenuously treating lesions of this character, particularly when the diagnosis is uncertain. The prognosis for cure is remote, but definite palliation has been obtained, for the patient has already lived over a year since the termination of radiotherapy.

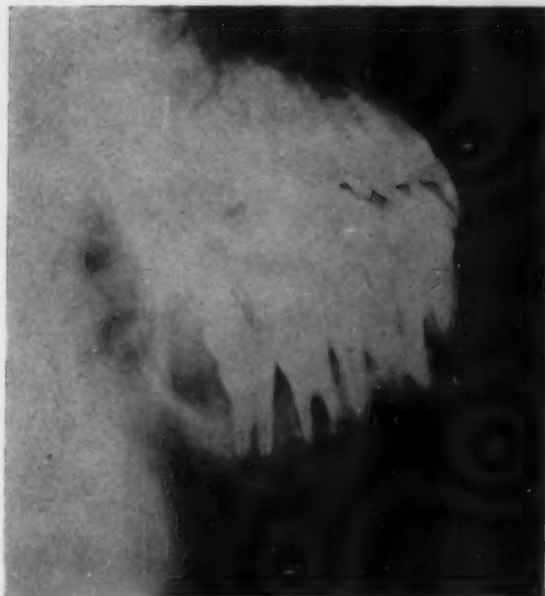


Fig. 75.

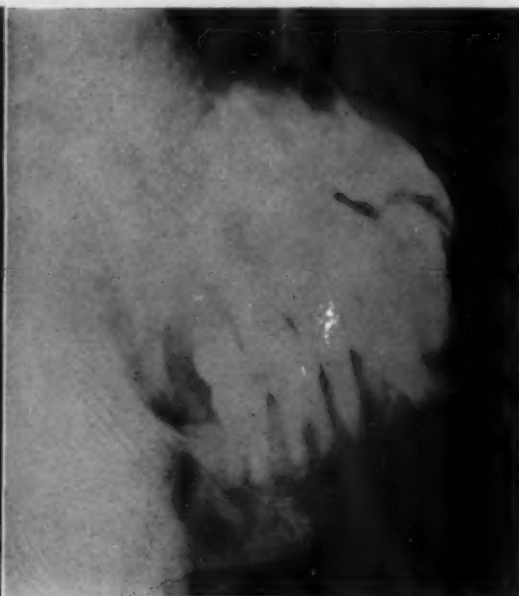


Fig. 77.

Fig. 75.—Oblique roentgenogram of left mandible showing radiolucency of body and alveolar absorption around teeth.

Fig. 77.—Oblique view of mandible showing calcification following radiotherapy.

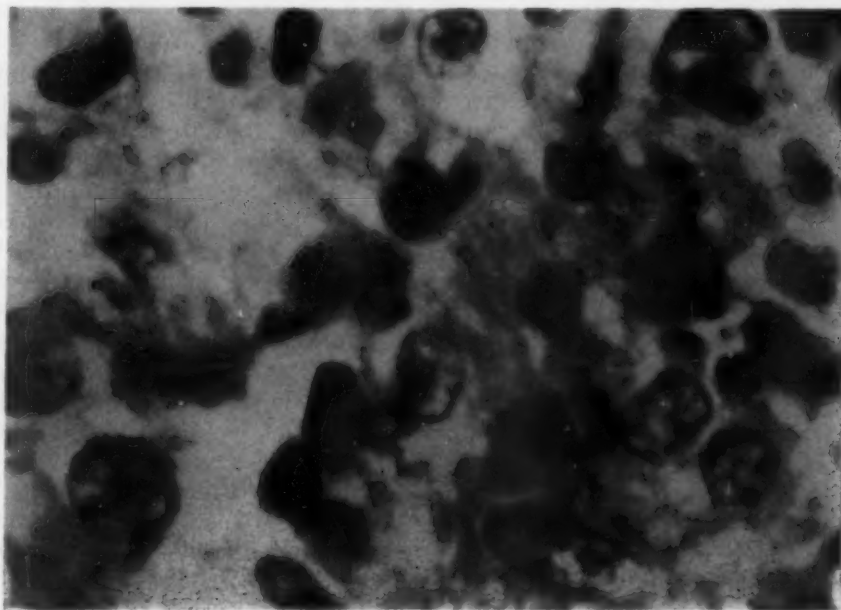


Fig. 76.—Photomicrograph (high-power). Note absence of osteoid with cells having indistinct cytoplasmic outlines with large nuclei and fine nucleoli.

Case 23, M. L., EFSCH No. 1428

Metastatic Ewing's Sarcoma of the Mandible

On Aug. 17, 1939, this 25-year-old woman came to the hospital complaining of swelling in the left arm. In 1936 the patient fell on ice and injured this arm which became painful and swollen below the elbow. There was accompanying fever. The clinical and roentgenologic diagnosis was osteomyelitis of the radius, for which, in February, 1939, a partial resection of the radius was done elsewhere.



Fig. 78.



Fig. 79.

Fig. 78.—Roentgenographic study of the left mandible showing radiolucent area in region of inferior dental canal.

Fig. 79.—Roentgenographic study of the right mandible showing radiolucent area in inferior border of mandible and molar area.

Examination showed a well-developed, well-nourished woman with a markedly prominent left antrum. The outer upper portion was tender and the overlying skin indurated with suggestive fluctuation. A roentgenogram taken on August 23 showed an area of destruction involving the lateral wall of the left orbit and the adjacent portion of the malar body. There was considerable soft-tissue tumefaction associated with this destruction, with fine parallel linear shadows extending transversely outward into soft tissue from the involved portion of the zygoma. The frontal and ethmoid sinuses were not remarkable, but both maxillary sinuses were slightly hazy. There were several ill-defined areas of bone rarefaction in the central portion of the parietal bone and in the vertical portion of the frontal bone. An x-ray of the forearm showed a soft-tissue tumefaction of the proximal portion. The proximal one-third of the radius had been amputated and the remaining bone showed poorly-defined, dense areas with some periosteal proliferation. Radiotherapy was directed to the skull and the forearm, which resulted in considerable alleviation of the pain and swelling.

In October, 1940, an x-ray of the mandible showed increased density of the posterior aspect of the left mandibular body. There was a well-defined, punched-out zone of structural bone loss in the region of the inferior aspect of the body of the left mandible. An irregular area of structural bone loss was present in the central aspect of the right mandibular canal (Figs. 78 and 79).

Consecutive bone lesions continued to develop (humerus, scapula, clavicle, left femur, lumbar vertebrae, and pubic rami) and temporarily but dramati-

cally responded to radiotherapy. The patient died on May 14, 1941, about five years after the first symptom.

Comment.—A Ewing's sarcoma rarely appears in a patient over 20 years of age.² It arises within the marrow cavity and the shaft of bones and produces symptoms which initially suggest osteomyelitis. As the disease progresses, unlike all other bone tumors, it frequently metastasizes to other bones.³ This particular case showed rather striking secondary involvement of the mandible, which produced radiologic changes. Radiotherapy produces a dramatic effect in relieving pain which invariably accompanies metastases. This pain may appear with increased sedimentation time before roentgenologic changes become apparent and there should be no hesitation in treating such areas.⁴ The clinical course of this case was typical but the duration was much longer than usual.

III. CARCINOMA OF THE BUCCAL MUCOSA

Introduction.—Carcinoma of the buccal mucosa makes up about 10 per cent of all intraoral neoplasms. It is primarily a disease of old age, about 90 per cent of the cases occurring in men. The midportion of the cheek at the occlusal level of the teeth is the most common site.

These carcinomas are usually quite well differentiated and to a great extent they parallel lip lesions in their histology. In Martin's series of 99 cases with a nine-month average duration of symptoms before admission, 56 patients had no palpable nodes on admission.⁵ During the entire time that the patients were followed, 51 (and not more than 56) developed metastases and in only 11 instances did the disease extend beyond the submaxillary lymph nodes.

This is one tumor in which chronic irritation due to sharp or broken teeth, poorly fitting dental appliances, syphilis, and tobacco may be implicated. In Martin's series, 25 per cent had evidence of dental irritation. Leucoplakia as a precursor also appears in about 20 per cent of the instances.

The differential diagnosis is usually not difficult. Tuberculous ulceration usually occurs on the tongue, but may be present anywhere in the oral cavity. However, when tuberculosis is present there are invariably extensive lesions present in the lungs. A piled-up leucoplakic area associated with infection may be suspicious of malignancy. Syphilis may also clinically look like tumor, but in all these instances biopsy will give the true diagnosis.

As a preliminary measure to therapy, all teeth on the affected side should be extracted whether that therapy is to be radiotherapeutic or surgical.^{6, 7} Irritative factors are thus removed and oral cleanliness is more easily retained. Radiotherapy to carcinoma of the buccal mucosa will quite naturally result in a certain amount of avascularization to the adjacent alveolar ridges with subsequent caries in the teeth on the irradiated side. This degree of caries is marked by a generalized cupping of the occlusal surfaces of the teeth and an abundance of cervical decay.⁸ This cervical destruction rapidly erodes the neck of the tooth and, unless molested, eventually amputates the crown of the tooth. Any surgical interference at this time to remove the residual roots will inevitably result in necrosis of the jawbone. Preliminary extraction of the teeth, particularly on the affected side, is a safeguard against future *injudicious extractions with subsequent radionecrosis of the irradiated jaw*.

Case 24, A. R., EFSCH No. 6365

A 58-year-old white man was admitted to this hospital in May, 1944. Two years previously, some white patches on the left buccal mucosa had appeared

and six months prior to admission a small scabbed sore developed on the vermillion border of the lower lip near the left commissure. A local physician told the patient that the lesion was probably cancer, but the patient was reluctant to believe this and treated himself with boric acid and glycerin. The growth continued to increase gradually in size.

Examination of the left buccal mucosa showed an exophytic ulcerated growth measuring 3 by 5 by 1 cm. This lesion extended from the left commissure of the mouth posteriorly and inferiorly to within 0.5 cm. of the buccogingival gutter (Fig. 80). On the left lower lip near the angle of the mouth there was a firm, slightly raised area measuring 0.7 cm., which was almost but not quite continuous with the buccal mucosa lesion. There was a single left submaxillary lymph node which was soft, slightly tender, and measured 1 cm. in diameter.



Fig. 80.—Photograph showing carcinoma of the buccal mucosa and right commissure of mouth with submucosal extension.

Biopsy specimens were taken from the lesion of the lip and the buccal mucosa.

Microscopic Diagnosis.—Oral cavity, lip, lower: Epidermoid carcinoma, Grade II, invasive. Primary tumor arose from the buccal mucosa.

X-ray studies of the chest were negative for evidence of parenchymal disease.

A total of 5,100 r. of x-ray therapy was given in thirty-three days with the following factors: 200 kv., 15 ma., 50 cm. TSD, 1 mm. copper and 1 mm. aluminum through one 6 by 5 cm. field. The hospital course was essentially uneventful except for the expected epithelitis and epidermitis which responded nicely to mouthwash and dressings and were almost healed at the time of a discharge in July, 1944.

This patient was readmitted to the hospital in August, 1944. Persistent carcinoma was present in the irradiated area and it was felt that further radiotherapy would be useless.

On August 11, under endotracheal ether anesthesia, an incision was made from a point 1.5 cm. from the left buccal mucosa on the upper lip, curved upward toward the malar eminence and posteriorly toward the region of the masseter muscle, inferiorly toward the area of the facial vessels, and then anteriorly to a point 1.5 cm. from the commissure of the lower lip. The commissure was reconstructed in layers by suturing the mucosa with fine catgut and the skin with cotton. The posterior aspect of the defect was closed anteriorly as far as

possible by the same method. There was difficulty in approximating the mucosa and for this reason it was elevated from the alveolar ridge by means of a periosteal elevator. The superior and inferior aspects of the defects were then closed with the exception of a small area in the center of the x-shaped closure. This defect was packed with zinc peroxide.

The postoperative course was uneventful. At the time of discharge the small defect in the left cheek was healing nicely.

The patient was readmitted to the hospital October 3 for a prophylactic left neck dissection, but aspiration biopsies of two submaxillary lymph nodes revealed metastatic disease on both the right and left. Because of this finding, palliative radiotherapy was given instead to the right and the left submaxillary regions but with no visible or subjective change. The palpable nodes in both cervical regions had not regressed at all at the time of discharge October 14. The patient died March 4, 1945, at home, ten months from the date of first admission.

Comment.—This was a very aggressive carcinoma of the buccal mucosa which locally recurred about one month after apparent adequate radiotherapy. In spite of radical excision, metastases appeared in both sides of the neck and the patient died about sixteen months after the first symptom.

Case 25, H. T., EFSCH No. 7245

This 61-year-old white man was first seen in the clinic on Feb. 19, 1945. Five years before, a small growth on the left buccal mucosa appeared but was attributed to rough teeth. All the teeth were extracted at that time, but the lesion slowly increased in size. Five months ago it began to grow rapidly and gave the appearance of a massive swelling in the left submaxillary space. There had been no treatment, no loss of weight, and no pain.

Examination revealed a massive adenopathy measuring 10 by 6 cm. occupying the entire left submaxillary space, which, at one point in its posterior-inferior aspect, was soft and fluctuant with reddened overlying skin. There was marked deformity of the face. Immediately inferior to the left commissure of the mouth was an infected draining lesion measuring 1.5 by 1.5 cm. There was a definite puckering of the left commissure extending inferiorly to the mass in the submaxillary space (Fig. 81).

Examination of the oral cavity revealed a large, exophytic lesion which was superficially ulcerated and extended from the left commissure of the mouth posteriorly to the left angle of the mandible on the substance of the left buccal mucosa. In its anterior third it was not attached to the alveolar ridge but, in its posterior two-thirds, appeared to become a part of the alveolar ridge and buccogingival gutter. It extended superiorly to the occlusal level of the upper alveolar ridge and measured 6 by 5 by 5 cm. A biopsy of the lesion revealed an epidermoid carcinoma, Grade I.

An oblique roentgenographic view of the mandible showed a small area of cupping of the posterior alveolar margin of the left mandibular body, possibly secondary to tumor invasion (Fig. 82).

On February 23 under endotracheal ether anesthesia, wide excision of the lesion was performed.

Microscopic Description.—Examination of the specimen showed that it had been adequately excised. The tumor was a well-differentiated squamous carcinoma which had grown out into the loose tissue. It had metastasized to a submaxillary lymph node and to four upper cervical lymph nodes. There was no evidence of mandibular bone invasion although the tumor was growing close to its surface.

Microscopic Diagnosis.—Oral cavity, buccal mucosa: Epidermoid carcinoma, Grade I, with metastases to 4 out of 16 lymph nodes (Figs. 83 and 84).

The postoperative course was uneventful and the patient was discharged from the hospital on the seventeenth postoperative day. When last seen on June 13, there was no evidence of local recurrence.

Comment.—This was a very extensive carcinoma of the buccal mucosa. Like most tumors in this region it was well differentiated, but unlike them it had metastasized to some of the upper cervical as well as the submaxillary lymph nodes. Such a radical surgical procedure in this instance was justified because of the well-differentiated character of the tumor with its tendency to remain

Fig. 81.



Fig. 82.

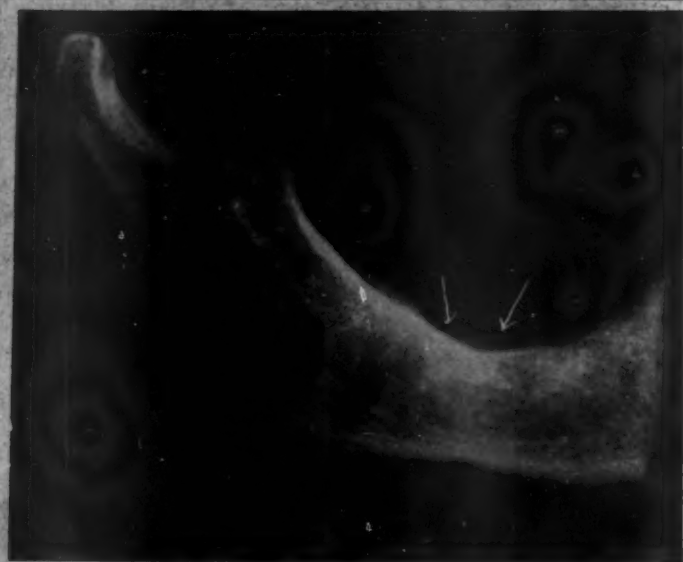
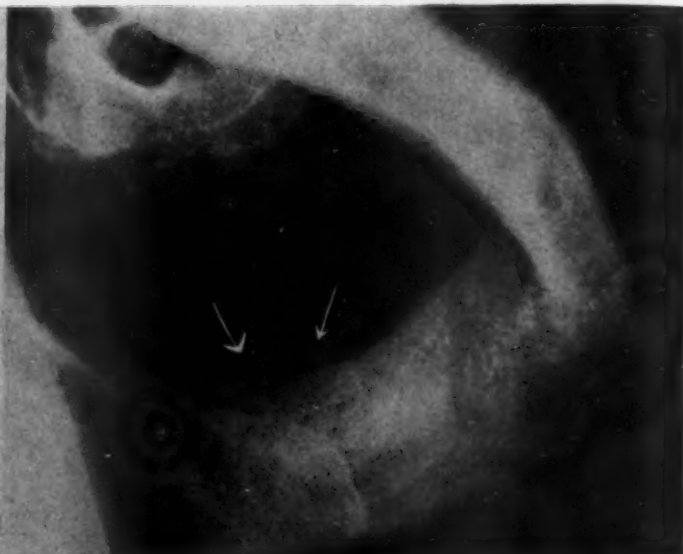


Fig. 83.

Fig. 84.

Fig. 81.—Photograph (full-face) showing metastatic mass in left submaxillary space and draining fistula, inferior angle of left commissure of mouth.

Fig. 82.—Roentgenographic study of left mandible showing slight loss of structure in alveolar portion, left second molar area.

Fig. 83.—Photograph of surgical specimen showing left mandible, tumor mass, and upper neck nodes.

Fig. 84.—Roentgenographic study of resected mandible.

localized. Radiation therapy to this tumor, because of its size, would probably have been rather unsatisfactory.

Carcinoma of the buccal mucosa has a better prognosis than is generally realized. Because of its usual well-differentiated character, it may be cured in a fairly high percentage of instances. In 99 consecutive cases treated by Martin, 28 were free from disease from five to eight years.⁵ In 9 of these 28 cases, metastases were present at some time during the course of the disease. Martin recommends treatment of the local lesion by radiotherapy and advises against routine prophylactic neck dissection because of the small percentage of metastases. It seems logical, however, that some individualization should be observed. If an originally undifferentiated tumor of the buccal mucosa has been controlled, a prophylactic radical neck dissection should be done.

IV. SALIVARY GLAND TUMORS

Case 26, M. K., EFSCH No. 6683

This 56-year-old white woman was first seen in the clinic in August, 1944, complaining of a lump in the left neck which had been present for approximately three years. This growth had gradually increased in size, and one year previous to our examination the local physician had surgically excised it. About two months later, another mass appeared in the same area and gradually increased in size.

Examination revealed an enlarged fluctuant area measuring 2 by 3 cm., located above the left parotid region, extending from the maxillary prominence downward to about the level of the thyroid cartilage (Fig. 85). Immediately inferior to this mass was an enlarged fluctuant node measuring 1.5 cm. in diameter. There was a left peripheral facial paralysis which developed after the first excision.

Aspiration biopsies of the parotid mass and the preauricular and supraclavicular lymph nodes were done.

Microscopic Diagnosis.—Primary malignant mixed tumor of the salivary gland with metastases to the cervical lymph nodes (Fig. 86).

Roentgenogram examination of the chest was negative for evidence of pulmonary metastases.

Because of the extent of the disease and the poor general condition of the patient, x-ray therapy to the left parotid and left cervical regions was given from September 5 to October 12 with the following factors: 200 kv., 15 ma., 60 cm. TSD, 1 mm. copper and 1 mm. aluminum over one 8 by 10 cm. and one 6 by 8 cm. field. At the time of discharge October 12, the mass in the left parotid region and the left cervical node had almost disappeared.

In January, 1945, the tumor of the parotid was entirely gone (Fig. 87). There was a nodule in the left upper cervical region measuring 1.5 cm. in diameter and another of the same dimension in the lower cervical region. Both of these nodules were immediately below the skin and were freely movable. X-ray examination at this time revealed no evidence of pulmonary metastases.

This patient was last seen May 23, 1945. At that time the parotid nodule had entirely disappeared. However, there was a palpable mass measuring 1 by 1 cm. in the left upper cervical region immediately inferior to the angle of the mandible. A return appointment in four months was given.

Comment.—Aspiration biopsy was a quick method for making a pathologic diagnosis. The course of this malignant salivary gland tumor was typical. In 82 cases of malignant salivary gland tumors reported by Ahlbom,⁹ 16 (or 20 per cent) had lymph node metastases on admission and 11 additional cases later developed metastases. Metastases to lungs and other organs probably occur in a much higher percentage than is generally believed, for the number of post-mortem examinations on these cases is few. Ahlbom stated that in 23 out of 63



Fig. 85.



Fig. 87.

Fig. 85.—Photograph showing tumor mass in left parotid region with tense overlying skin.
Fig. 87.—Photograph showing complete regression of mass following radiotherapy.

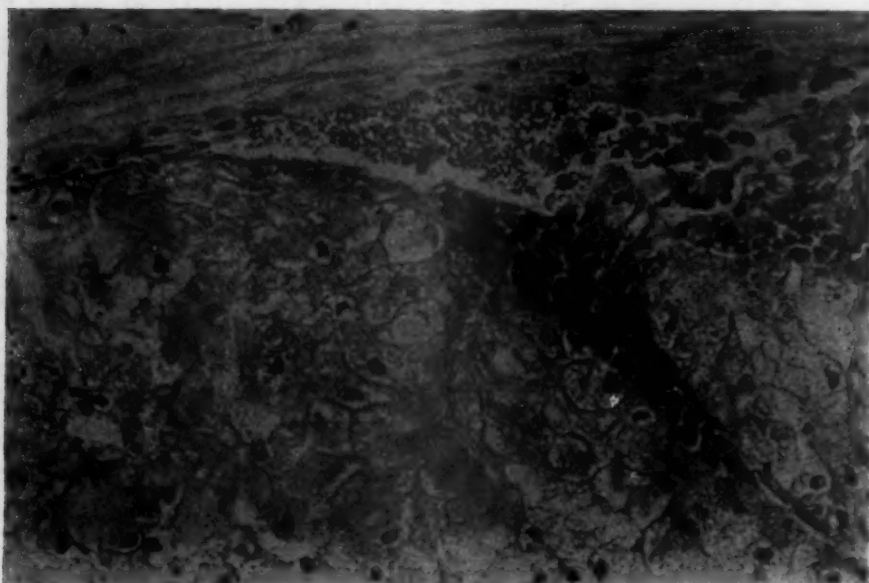


Fig. 86.—Photomicrograph (low-power). The cervical lymph node is completely replaced by well-differentiated tumor.

cases metastases could have been present and were suspected but no proof was obtainable. It is his belief that distant metastases are more common than lymph node involvement. For this reason it is our policy to take routine roentgenograms of the chest on all patients presenting salivary gland tumors.

When surgery is contraindicated because of far-advanced disease or because the patient is a poor surgical risk, radiotherapy is definitely indicated. This therapy should be given with the hope of sterilization although this probably rarely occurs. However, definite palliation usually results and the tumor may apparently disappear (as in this patient). This is followed by a period of quiescence which may last several years before recurrence takes place.

Case 27, C. P., EFSCH No. 2406

This 89-year-old white woman was first seen in the clinic in January, 1941, complaining of a swelling of the face and hard palate of three years' duration. Due to a lack of cooperation on the part of the patient plus a marked speech impairment, the history was very difficult to obtain.

Examination revealed the right side of the face to be distorted by a mass which involved the region of the maxillary sinus, but the right eye was not affected. There was a necrotic, superficially ulcerated mass protruding from the mouth, and intraorally the entire substance of the left hard palate was involved sufficiently to prevent closure of the mouth (Fig. 88).

Roentgenogram examination showed a massive soft tumefaction extending anteriorly and laterally from the right antral region mesially beyond the nasal septum and posteriorly into the nasopharynx. The mass presented several small, irregular linear areas of increased density in its anterior portion. It had destroyed by erosion the mesial and lateral walls of the antrum, the left lateral aspect of the palate, a portion of the nasal septum, and the greater portion of the left malar bone. The mouth was edentulous with the exception of a lower third molar on the right and a root peg in the maxilla. The mandible was markedly atrophic (Fig. 89).

A biopsy of the soft palate was performed on January 12.

Microscopic Description.—The tumor showed a tendency to form acini which were well differentiated. This was a rather slow-growing tumor of low malignancy, of salivary gland origin.

Microscopic Diagnosis.—Oral cavity: Mixed tumor, questionably malignant (Fig. 90).

The patient was very uncooperative and requested dismissal from the hospital on January 15, but returned on January 30 in poor general condition and with facial pain. A total of 1750 r. of palliative therapy was given to the left antral area, but the patient rapidly became comatose and expired on Feb. 4, 1941.

The post-mortem examination showed extensive local invasion of the antrum and floor of the orbit, but there was no extension down the pharynx or evidence of metastases elsewhere. The immediate cause of death was aspiration pneumonia.

Case 28, E. W., EFSCH No. 2349

This 66-year-old white woman was first seen in the clinic, Dec. 29, 1940. In 1928 an upper tooth was extracted and the patient was told at that time that the root of this tooth adjoined a tumor mass. Five years later in 1933 the growth was surgically removed from the upper lip, but recurred and was re-excised in 1935. There had been a gradual enlargement of the right cheek with some lacrimation and gradual loss of sight in the right eye. Approximately one year prior to our examination, x-ray therapy was given to the right cheek.

Examination revealed a firm hard mass of the right buccal mucosa involving the upper half of the right cheek. There was a diffuse deformity over the right antrum and the conjunctiva of the right eye was injected. There was a right facial paralysis with apparent anesthesia of the third division of the fifth nerve

on the right side. The submaxillary nodes were bilaterally enlarged and hard. There was a deep submucosal mass growing on the right side of the nasopharynx.

Roentgenogram examination showed all the paranasal sinuses to be hazy. The walls of the right antrum were thickened and their inner margins were fuzzy and ill defined. The density of the malar body at that time was irregular and there was a suggestion of a minimal amount of structural bone loss. The mouth

Fig. 88.



Fig. 89.

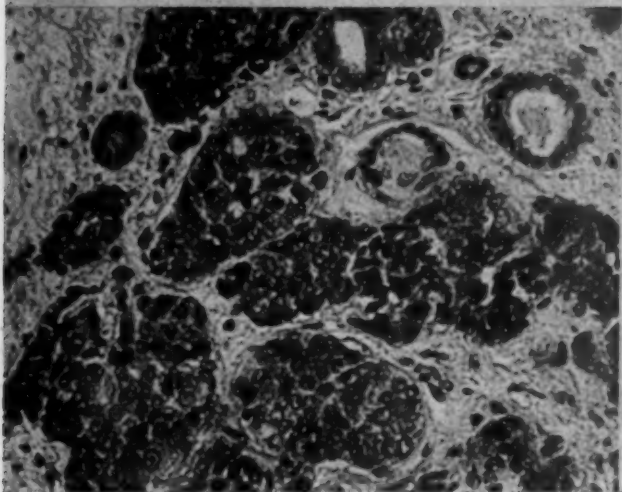
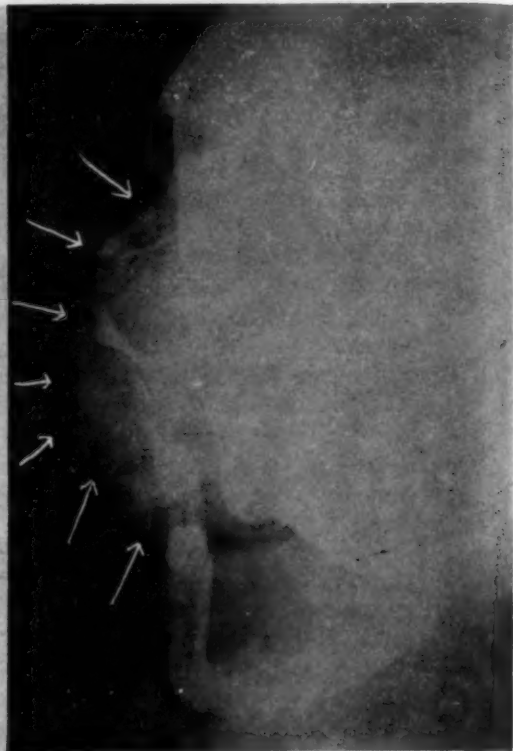


Fig. 90.

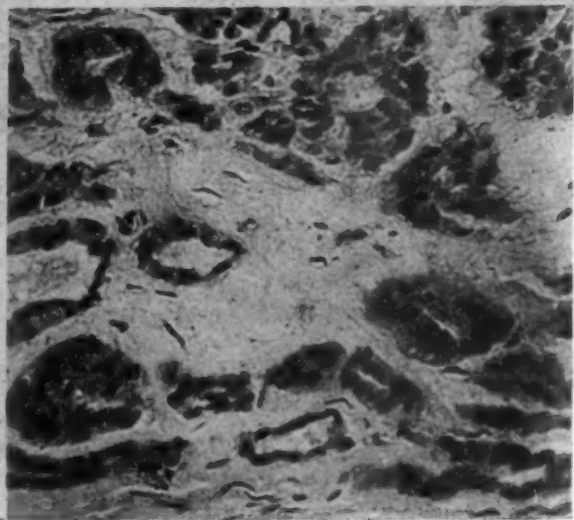


Fig. 91.

Fig. 88.—Photograph showing tumor mass in upper jaw.

Fig. 89.—Roentgenographic study showing extension of tumor, destruction of lateral and mesial upper right maxilla and antrum.

Fig. 90.—Photomicrograph (low-power). Salivary gland tumor showing tendency toward acinar formation.

Fig. 91.—Photomicrograph (low-power). This almost exactly resembles the tumor represented in Fig. 90.

was edentulous. There was a considerable amount of atrophy of the mandible. The foramen ovale on the right was markedly enlarged. There was evidence of bone erosion in its adjacent dependent portion which communicated with the foramen proper. This examination revealed a confluence of the foramen ovale.

A biopsy of the buccal mucosa was taken.

Microscopic Description.—Some of the cells were arranged in a definite glandular formation. In the glands the nuclei were at the base of the cells and were oval and even-staining with definite polarity. This was a slowly-growing tumor and parts of it resembled the adenoides cystica type of basal cell carcinoma. This tumor evidently arose from salivary gland and was a mixed tumor which could be expected to invade contiguous tissue directly.

Microscopic Diagnosis.—Oral cavity, buccal mucosa: Mixed tumor, questionably malignant (Fig. 91).

Radiotherapy was given to the lesion from January 17 to February 4 with some decrease in cranial pain and some regression of the mass over the right antral area. The patient was discharged February 5.

Re-examination on March 19 revealed the tumor mass in the right infra-orbital region to be considerably regressed. The cheek was soft and pliable and there had been remarkable regression of pain.

On May 16 there was no reactivation of the growth other than a very slight increased thickening of the right cheek. There was severe pain in the right eye-ball, however, and on July 19 a posterior root resection of the fifth nerve was carried out. The entire posterior root was sectioned approximal to the ganglion and the ganglion removed.

Microscopic Diagnosis.—Nerve (fifth): Mixed tumor, malignant, invasive.

The result of this procedure was very satisfactory and the patient obtained much relief from pain.

On August 29 there was severe conjunctivitis of the right eye and pain in the right maxillary region. The patient was last seen in October still complaining of pain in the right side of the face. Tumor was still present in the lip and the right nasolabial fold. The right eye was markedly exophthalmic. We did not believe that any further radiation therapy would be of value. Death occurred at home in December, 1942, two years after the first admission.

Comment.—These two tumors arose from the salivary glands of the oral cavity and showed similar pathology. Salivary gland tumors tend to invade contiguous tissues and locally to invade the palate. They may eventually force their way into the nasal cavity and accessory sinuses and may also extend down the hypopharynx. At times, as in the second case, they invade large nerves. These tumors may eventually produce impairment in eating, speaking, and respiration, which in turn cause rapid weight loss. This is frequently followed by infection and terminal aspiration pneumonia. When the growth is too large to remove by surgery, radiotherapy may give real palliation with relief of pain and diminution in the size of the tumor.

V. EXTENSIVE AMELOBLASTOMA

Case 29, F. F., EFSCH No. 9654

This 69-year-old Negro was first seen in the clinic in January, 1943, complaining of a swelling in the left mandible. In 1933 a swelling appeared around the left lower third molar with a gradual loosening of the first and second molars. The patient sought consultation and had these teeth removed. In 1937 the first and second premolars in the left mandible were extracted and a small cyst removed. Shortly after this surgical procedure, there was a gradual enlargement of the left mandible but no pain.

Examination revealed a large fluctuant mass measuring 5.5 by 4 cm. covering the alveolar aspect of the left mandible which was edentulous from the left

lower canine to the third molar. It extended superiorly to occlude with the left maxillary premolars and molars. The overlying mucosa was normal with no evidence of superficial ulceration. There was a definite facial deformity but no palpable cervical adenopathy. An aspiration biopsy was performed.

Microscopic Diagnosis.—Ameloblastoma of the left mandible (Fig. 92).

X-ray studies showed a definite multilocular cystic degeneration extending from the left molar area across the symphysis to the right second molar area (Figs. 93, 94, and 95).

Fig. 93.



Fig. 94.



Fig. 95.

Fig. 92.

Fig. 92.—Photomicrograph (low-power). Ameloblastoma (glandular type).

Fig. 93.—Roentgenographic study of left mandible showing multilocular cystic degeneration.

Fig. 94.—Roentgenographic study of right mandible showing multilocular cystic degeneration.

Fig. 95.—Posteroanterior view showing extension of radiolucency across symphysis of mandible.

The treatment of choice would have been a wide excision of the mass, but when the patient realized that this excision would also include a hemisection of the lower jaw, this treatment was refused. Consequently only a partial removal of the cystic areas was performed with only that portion of the mandible removed which could be picked up with the rongeurs. The postoperative course was uneventful.

In April, 1945, there was definite evidence of recurrence in the left mandible with extension across the midline of the symphysis to the body of the right mandible. The patient still refused any radical procedures. There had been a steady loss of weight for the past year, but x-ray studies of the chest failed to reveal any evidence of parenchymal disease.

Comment.—This patient had the typical long course of an ameloblastoma with symptoms lasting at least twelve years.¹⁰ It had, however, extended across the symphysis of the mandible, which is distinctly unusual. Probably, radical surgery at this point is still possible, although extensive plastic repair would be necessary. With ulcerated ameloblastomas it is worth while to take roentgenograms of the lungs, for very rarely metastases can take place.¹¹

VI. EPIDERMOID CARCINOMA OF THE ALVEOLAR RIDGE WITH RADIONECROSIS OF THE MANDIBLE

Case 30, W. S., EFSCH No. 6017

This 70-year-old man was first seen in the clinic in January, 1944, stating that in 1942 he noticed a small growth on the lower jaw. It looked like a pimple but increased in size until it extended over into the substance of the left cheek, causing some difficulty in opening the mouth. In April, 1943, a biopsy of the lesion was taken.

Microscopic Diagnosis.—Epidermoid carcinoma, Grade II. Radium therapy was given elsewhere with slight regression of the lesion but in the fall of 1943 a small persistent ulcer appeared on the previously treated side.

Examination revealed an ulcerated area measuring 2 by 0.7 cm. along the left inferior alveolar ridge. The base of this ulcer was composed of necrotic bone. There was no trismus and the tissues of the submaxillary region were woody. There were no palpable cervical nodes.

Roentgenogram examination of both mandibles revealed nothing but a small zone of structural bone loss in the alveolar aspect of the left mandibular body (Fig. 96).

A biopsy of the lower gum showed only chronic inflammation. This lesion seemed to represent radionecrosis and the patient was advised to maintain strict oral hygiene and return in two months.

The patient was not seen again until September, 1944, when examination revealed the same ulcerated area along the left inferior alveolar border with very obvious areas of disease present. This was considered to be recurrent carcinoma of the left inferior alveolar ridge with bone involvement.

Roentgenograms revealed a marked increase of destructive bone change involving the left mandibular body. The midportion of the ramus was included and a fracture was present in the posterior body (Fig. 97).

A second biopsy of the gum showed considerable necrosis with a few small spicules of bone, but no tumor was identified.

Microscopic Diagnosis.—Radiation effect.

In spite of repeated biopsies with no evidence of carcinoma, it was generally agreed that there was a possibility that residual carcinoma of the mandible was present. A resection of the left mandible was done on September 26.

Microscopic Description.—Marked fibrosis, chronic inflammation, and replacement of muscle by connective tissue was observed. Calcification of the walls of the large blood vessels was seen. The submaxillary gland showed marked interacinar and interlobular fibrosis. There was no evidence of tumor in the sections. Some of the blood vessels were completely obliterated and the

nerves showed degenerative changes. Some of the muscle was replaced by fibrosis.

The original biopsy taken elsewhere in April, 1943, showed that this was a definite carcinoma. Multiple biopsies before the operation showed no evidence of carcinoma and all sections taken of the operative specimen showed only the effects of radiation and infection.

Fig. 96.

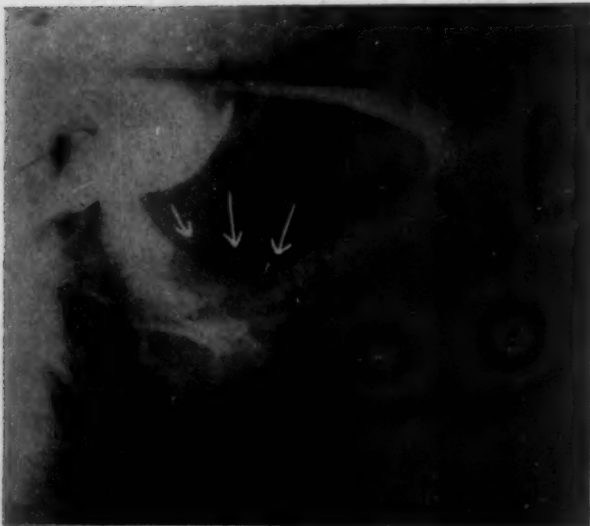


Fig. 97.

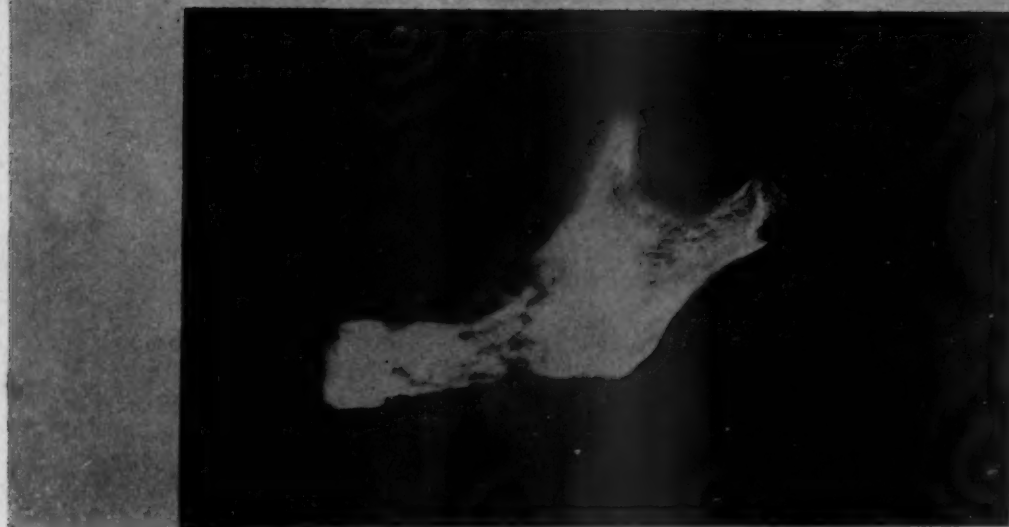
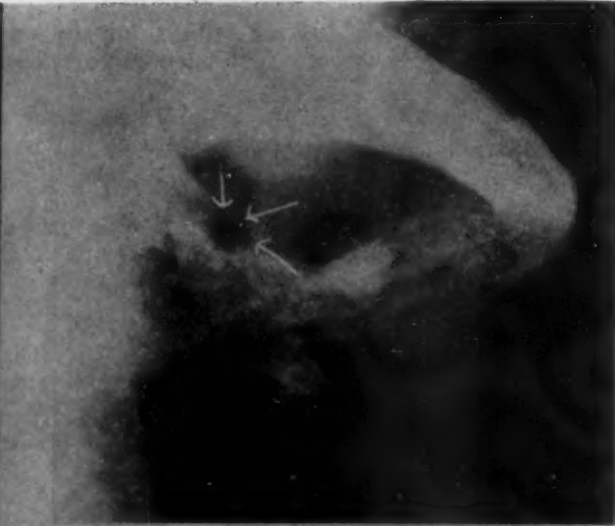


Fig. 98.

Fig. 96.—Roentgenographic examination of left mandible showing slight area of radiolucency, alveolar ridge in the area of the third molar.

Fig. 97.—Roentgenographic study of left mandible showing an area of destruction with pathologic fracture at the angle.

Fig. 98.—Photograph showing x-ray study of surgical specimen of left mandible.

Microscopic Diagnosis.—Oral cavity, gum (mandible): Radiation effect. Bone, mandible: Radiation effect. Muscle: Radiation effect. Salivary gland, submaxillary: Radiation effect (Fig. 98).

The postoperative course was complicated by aspiration pneumonia which was treated by sulfonamides and penicillin, and a mild cardiac decompensation which was treated with digitalis. The nonprotein nitrogen increased to 50 mg. per cent and there was a high urinary residual which subsided. Shortly

afterward, a *B. pyocyaneus* septicemia developed and was treated by 5 million units of penicillin over a period of four weeks. The blood stream was eventually sterilized and the patient was discharged in November with a small salivary fistula on the left side of the face resulting from a minimal wound infection. It was felt that this fistula would eventually close entirely or become smaller.

A report from the county nurse in January, 1945, stated that the patient had expired twenty months after the first treatment.

Comment.—Radionecrosis of the mandible is an unfortunate complication which may occur following radiotherapy. This necrosis may be unfavorably influenced by the presence of the expected concomitant infection. Because of these progressive changes, it may be necessary, as in this case, to resect the mandible. The oral hygiene in this case was foul and death undoubtedly was caused by aspiration necrotizing pneumonia.¹²

VII. HEMANGIOMA OF THE LIP

Case 31, L. E., EFSCH No. 2671

This 19-year-old Negro woman was first seen in the clinic, April, 1941, complaining of a swelling of the upper lip present since childhood. This growth had gradually increased in size but there was no history of pain or discomfort other than difficulty in speech due to the prominent size of the lip lesion.

Examination revealed a globular swelling measuring 2 by 2.5 cm. in the midportion of the upper lip. It extended from the vermilion border to the labial mucous membrane at the labiokingival gutter and laterally to the left commissure (Fig. 99).

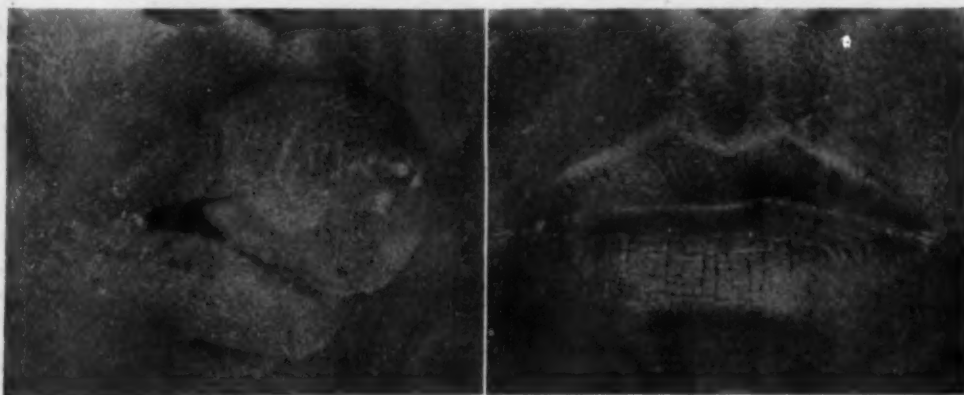


Fig. 99.

Fig. 100.

Fig. 99.—Photograph showing cavernous hemangioma, upper lip.

Fig. 100.—Photograph three years following excision.

On April 18, under endotracheal ether anesthesia an incision was made around the limits of the tumor on the labial mucosal side and the tumor was then dissected free of contiguous tissues. Near the labiokingival fornix there were two rather large vessels entering the tumor mass. These were clamped and ligated. The lip was reconstructed by replacing the labiomucosal flap with interrupted silk sutures. A pressure dressing was then applied and the patient was discharged April 29 with a return appointment for one month.

Microscopic Diagnosis.—Oral cavity, lip, upper: Cavernous hemangioma.

This patient was re-examined at intervals of six months until November, 1944, and at no time was there any evidence of recurrent or persistent hemangioma. The cosmetic results were satisfactory with only a barely visible scar on the mucous surface of the lip (Fig. 100). The patient was discharged November 28 and the case was closed.

Comment.—This was a bulky, deforming lesion for which sodium morrhuate did not seem practical because of the dangers of necrosis, the difficulties of completely eradicating it, and because it is time consuming. Surgery, however, was a rapid, effective method of completely excising the lesion, with an excellent cosmetic result.

VIII. LESIONS OF THE LOWER LIP

Introduction.—The following lip cases are presented to illustrate the type of lesions found on the lip and the variance of therapy. Carcinoma of the lip is the most common lesion of the oral cavity and the type of treatment indicated is entirely dependent upon the size of the area to be treated, its grade, location, and its previous therapy. Small, well-circumscribed lesions of the lip (less than 1 cm.) offer a good cosmetic and therapeutic result by simple v-excision or radiotherapy. Larger lesions (1 to 2 cm.) can be treated by v-excision if the lip is relaxed so that a good cosmetic result may be obtained. Radiotherapy is also effective. The radium mold is also a satisfactory but time-consuming procedure which offers no advantages over v-excision or radiotherapy and is therefore not recommended. Larger lesions are best treated by radiotherapy. Surgical excision in these lesions would result in a nonfunctional lip and would necessitate plastic repair and require unnecessary hospitalization. Lesions previously treated by radiation or zinc chloride, which recur, will of necessity require surgery and may have to be followed by plastic repair.

Case 32, J. C., EFSCH No. 5160

Benign Lesion Simulating Carcinoma

This 72-year-old white man was first seen in the clinic in April, 1943, complaining of a lesion of the lower lip which had been present for three months.

Examination revealed a large, exophytic lesion measuring 3 by 2 by 2 cm. located in the midline and to the right of the lower lip (Figs. 101 and 102). A small submental node 1 cm. in diameter was present. A biopsy of the lesion was performed.

Microscopic Description.—Marked hyperkeratinization, hyperplasia, and chronic inflammation are present. Although the basement membrane is ragged due to inflammation, these changes do not suggest cancer.

Microscopic Diagnosis.—Oral cavity, lip, lower: Hyperkeratinization, hyperplasia, and chronic inflammation.

An aspiration biopsy of the submental node was reported negative.

Clinically this lesion suggested a low-grade carcinoma of the lip, and the patient received 4,750 r. of x-ray therapy through one 3 by 4 field, 100 kv., ¼ mm. copper in thirty-one days.

At the time of discharge on May 23, the tumor had entirely disappeared (Fig. 103). The submental node had shown no change in size.

This patient was re-examined at two-month intervals until December, 1943, at which time the submental node was slightly larger. A second aspiration biopsy of this submental adenopathy again failed to show any evidence of disease. The last examination was in December, 1944. The lip was soft and pliable and there was no palpable submental adenopathy or evidence of disease.

Comment.—In our experience, about 35 per cent of the lesions of the lower lip prove to be benign. In some instances the benign character of the lesions was clinically apparent when seen, and a diagnosis of leucoplakia or benign hyperkeratosis was made and confirmed. In rare instances syphilis was mistaken for carcinoma. However, the majority of the cases exactly simulated malignant disease, and microscopic examination alone revealed their benign character. It is true that practically all of these lesions were precancerous, such



Fig. 101.



Fig. 102.



Fig. 103.

Fig. 101.—Photograph (full-face) showing lesion, right lower lip, and midline.
Fig. 102.—Photograph (profile) showing lesion.
Fig. 103.—Photograph showing lip following radiotherapy.

as this one. Induration of the base of the lesion accompanied by tender, enlarged regional nodes may be merely an indication of inflammation. Benign lesions rarely attain the size of the one reported here. These lesions are best treated by v-excision or radiotherapy. Often the mouth hygiene is poor, and of course this should also be cared for.

Epidermoid Carcinoma Without Metastases

Case 33, G. H., EFSCH No. 4661

This 64-year-old white man came to the hospital in October, 1942, complaining of a growth on the left lower lip of three years' duration. A pipe had always been held at the site of this tumor.

Examination revealed a hard, irregular mass measuring 3 by 2.5 by 2.5 cm. on the left lower lip, involving the entire thickness of the lip and extending to the left commissure of the mouth (Figs. 104 and 105). A biopsy of the lesion was taken.

Microscopic Diagnosis.—Oral cavity, lip, lower: Epidermoid carcinoma, Grade I.

Radiotherapy was given from October 22 to November 4, and totaled 3,600 r. through a 4.5 by 3 cm. field using 150 kv., 10 ma., 30 cm. TSD with 3 mm. of aluminum.

The patient was re-examined every six months and was last seen in June, 1944, at which time there were no palpable nodes in the neck or evidence of persistent disease. The radiation defect was soft and pliable (Figs. 106 and 107).

Comment.—This was a typical, large, exophytic type of squamous carcinoma, well-differentiated in character and without evidence of metastases to the regional lymph nodes. Surgery would have resulted in deformity necessitating plastic repair. Roentgentherapy resulted in complete cure with little deformity, leaving a pliable, soft lower lip.

Case 34, A. A., EFSCH No. 4483

This 60-year-old white man was first examined in the clinic in August, 1942. About a year before, a small ulcerated area first appeared in the midline of the lower lip. This had slowly increased in size and periodically became ulcerated and bloody. A few weeks previous to examination, it started to grow quite rapidly. The patient had been a confirmed pipe smoker for many years, but was somewhat vague as to the portion of mouth in which the pipe was held.

Examination revealed a rough, superficially ulcerated lesion measuring 3.5 by 2.5 by 2 cm. in the midportion and left half of the lower lip. There were no palpable lymph nodes (Fig. 108). The remaining teeth were in very poor repair and the supporting mucosa was inflamed. A biopsy was done on July 8.

Microscopic Diagnosis.—Oral cavity, lip, lower: Epidermoid carcinoma, ungraded.

All the remaining teeth were extracted, and 2,700 r. of x-ray therapy was directed to the lesion with the following factors: 150 kv., 10 ma., 30 cm. TSD, 3 mm. aluminum, through one field 5 by 3 cm. The patient was discharged in October with a marked epithelitis but with some diminution in the size of the lesion.

Re-examination in November showed marked regression of the lesion with no signs of persistent or recurrent disease. The left submaxillary space presented a node 1 cm. in diameter, but the patient had to return home for business reasons and no further examination of it was possible at this time. In January, 1943, the node had not increased in size and there was no evidence of metastasis.

The patient was examined every six months and was last seen December, 1944, at which time there was no evidence of local recurrence nor any discernible cervical adenopathy. The cosmetic result was excellent, showing only a slight scar and achromia at the site of the lesion (Fig. 109).

Fig. 104.



Fig. 105.



Fig. 106.



Fig. 107.

Fig. 104.—Photograph (full-face) showing carcinoma of left lower lip.

Fig. 105.—Photograph (profile) showing carcinoma of lower lip.

Fig. 106.—Photograph (full-face) following radiotherapy, showing only a slight loss of substance with no noticeable deformity.

Fig. 107.—Photograph (profile) following radiotherapy, showing only a slight loss of substance with no noticeable deformity.

Comment.—Surgical removal of this lesion would have necessitated extensive plastic repair, whereas the cosmetic result following radiotherapy was excellent. In such large, obviously infected lesions, enlarged regional lymph nodes are commonly present but this enlargement is usually only inflammatory. If there is any doubt, aspiration biopsy should be performed. If a node develops after treatment, even if aspiration biopsy is negative, follow-up visits should be at closer time intervals. During radiotherapy the lower anterior teeth are protected by a lead shield placed between the lip and the labial portion of the teeth.



Fig. 108.

Fig. 108.—Photograph showing carcinoma of lower lip.



Fig. 109.

Fig. 109.—Photograph showing carcinoma of lower lip following radiotherapy.

Routine prophylactic neck dissections are not indicated in carcinoma of the lower lip, as only about 25 per cent develop any metastases. In a hypothetical series of 100 cases with carcinoma of the lower lip, if carcinoma in the regional lymph nodes is present at first examination (15 per cent), therapeutic upper neck dissection should be done. If metastases develop after treatment (10 per cent of the remaining 85 cases), they invariably appear only in the submental or submaxillary node areas (90 per cent). Because of this localization of spread, in most instances the disease can be completely removed by an upper rather than a complete neck dissection. If the primary tumor is a very undifferentiated carcinoma, then a radical neck dissection is indicated.

Case 35, J. O., EFSCH No. 3251

This 67-year-old white man was first seen in the clinic, September, 1941, complaining of an ulceration on the lower lip present for approximately three years. There had been no pain but for two months there had been drooling about this ulceration.

Examination revealed the entire lower lip to be replaced by dirty, foul-smelling, irregular, ulcerating, fungating growth which involved the alveolar mucosa of the mandible and the anterior floor of the mouth. It also extended laterally past the commissures (Figs. 110 and 111). A biopsy was made of the lesion.

Microscopic Diagnosis.—Epidermoid carcinoma, Grade I.

Roentgenogram examination showed an enormous soft-tissue tumefaction extending outward from the anterior aspect of the chin. There was a large

crater in the tumefaction which extended to the mandible and left laterally to the symphysis where there was an area of bone destruction.

Diagnosis.—Large soft-tissue tumor of the chin, with associated crater and bone destruction left laterally to the symphysis.

A total of 2,500 r. to two lateral fields each measuring 10 by 10 cm. was given, and the patient was discharged on October 4 with slight regression and noticeable improvement in the cleanliness of the lesion.

Fig. 110.



Fig. 111.

Fig. 110.—Photograph (profile) showing extensive carcinoma of lower lip and involvement of chin.

Fig. 111.—Photograph (full-face) showing extensive carcinoma of lower lip.

Re-examination in January, 1942, showed some regression of the tumor particularly at the commissures. In the middle of the chin the ulceration was somewhat deeper. A massive bilateral resection of the mandible, together with the soft tissues of the lower lip and the floor of the mouth, seemed to offer the only hope for cure. However, the patient was quite frail and had been bedfast for many weeks. These factors contraindicated radical surgery and the patient was discharged. He died at home on April 25, 1942.

Comment.—This represents the far-advanced carcinoma of the lower lip for which cure can only rarely be effected, either by extremely radical surgery

or by intensive radiotherapy. It is rather remarkable that definite evidence of lymph node metastases was lacking.

**Post-Zinc Chloride Recurrence of Epidermoid Carcinoma
Case 36, C. G., EFSCH No. 6643**

This 64-year-old white man was first examined in the clinic in August, 1944. About two years before, a small growth was first noticed on the lower lip. In December, 1943, the local physician applied generous portions of salve to the lesion but the results were so discouraging the patient refused to return for re-examination. In May, 1944, on the advice of some filling station operator, he applied a black paste to the lower lip and a large portion of the lip came off and the pain was severe.



Fig. 112.

Fig. 112.—Photograph showing recurrent carcinoma of lip following paste treatment received elsewhere.



Fig. 113.

Fig. 113.—Photograph showing lower lip following radiotherapy.

Examination revealed a widely ulcerated lesion of the vermilion border and skin of the lower lip, measuring 5 by 2 by 1.5 cm. It was covered with a black scab and presented raised borders (Fig. 112). There were no palpable nodes in the neck.

A biopsy was done.

Microscopic Diagnosis.—Epidermoid carcinoma, Grade I, post-paste recurrence.

A total of 4,700 r. through one 7 by 4 cm. field was given in thirty-four days with 200 kv., 15 ma., 50 cm. TSD, with a $\frac{1}{2}$ mm. copper and 1 mm. aluminum filtration.

At the time of discharge in September there was a radioepidermitis of the treated area with some diminution of the lesion.

The patient was examined at intervals of two months and was last seen in June, 1945. There was some loss of substance and some shortening of the lower

lip but there were no marked atrophy or sequelae and no evidence of local recurrence or cervical metastases (Fig. 113).

Comment.—Skin and lip lesions are frequently treated by some form of paste (often zinc chloride) before seen at this hospital. This paste indiscriminately destroys any tissue with which it comes in contact. Carcinoma frequently recurs in these relatively avascular areas, making treatment by radiation more difficult, and, by surgery, necessarily more extensive. However, the majority of these cases can be salvaged.¹³

Epidermoid Carcinoma With Metastases

Case 37, T. F., EFSCH No. 4109

A 61-year-old white man first came to the clinic in May, 1942, complaining of a hard mass attached to the right mandible. A small lesion developed on the left side of the mouth in August, 1935, but was apparently completely removed in May, 1936. In December, 1941, another lesion was removed by cautery from the left angle of the mouth. In March, 1942, a large hard mass inferior to the right mandible appeared, and x-ray therapy was given at an employer's hospital. This treatment resulted in little obvious improvement and the lump continued to increase in size.

Examination revealed a hard, painful mass measuring 7 by 5 by 4 cm., inferior to the right mandibular body and occupying the entire right submaxillary fossa (Fig. 114). The overlying skin was tense, shiny, and thin, and there was fluctuation immediately beneath it. There was a definite facial asymmetry. Intraoral examination revealed a small scar on the buccal mucosa just posterior to the right commissure and a palpable mass replacing the greater portion of the floor of the mouth on the right. The tongue protruded in the midline. An aspiration biopsy was performed.

Microscopic Diagnosis.—Lymph node, submaxillary: Epidermoid carcinoma, ungraded, metastatic. The primary tumor arose from the oral cavity, lower lip.

Radiologic examination revealed the mouth to be edentulous with no evidence of destructive bone changes in either mandible. There was a slight marginal irregularity along the alveolar aspect of the left mandibular body.

On May 26, under endotracheal ether anesthesia, an en bloc resection of the right mandible and a right radical neck dissection were done.

Operative Procedure.—Using endotherm current the gingival buccal mucosa was incised followed by incision of the gingival lingual mucosa. These incisions were bluntly deepened along the lateral and medial borders of the upper portion of the mass. A curvilinear incision was then made from well behind the jaw angle to the left mental trigon, and a vertical incision down the sternomastoid muscle was carried out from the midpoint of the curvilinear incision to the suprasternal notch. The sternomastoid muscle was divided about an inch above its sternal and clavicular attachments. The underlying internal jugular vein was identified, freed, clamped, and divided. Its distal end was ligated with a single medium black silk suture and the contents of the posterior triangle were dissected free. The flaps were freed to the midline posteriorly and to the posterior border of the sternomastoid muscle laterally. All fatty node-bearing tissue adjacent to the sternomastoid muscle and vein was stripped upward dividing the cervical roots as encountered. Just above the carotid bulb, the superior thyroid and laryngeal vessels were identified and the external carotid artery divided distal to these two vessels. The upper flap was now developed to a point well up on the masseter muscle. The facial vessels were divided and ligated. The masseter muscle was divided just above the prevascular and the postvascular nodes. The insertion of the anterior belly of the digastric muscle into the mandible was divided and the contents of the left and right submental triangles and the submaxillary space dissected downward and laterally far enough to permit good access to the mandible. The neck and oral cavity wounds were now made continuous by blunt dissection. A Gigli saw was passed around the mandible at about the position of the mental tubercle and the mandible was

divided. In a similar fashion the saw was passed around the ascending ramus of the mandible well above the angle and the mandible again divided. This allowed considerable mobility and permitted stripping the mylohyoid muscle, the mandible, and the mass from the lingual nerve, lingual muscles, and hypoglossal nerve. This allowed the entire contents of the submaxillary triangle to be dissected downward. The facial artery was again ligated and divided, the tip of the parotid gland was cut across and the dissection continued downward and posteriorly to meet the lower dissection, which freed the entire specimen. The portion of remaining mandible was now grasped, the insertion of the external pterygoid and temporalis muscles divided, and the fragment disarticulated. Some bleeding was encountered following disarticulation which could only be controlled by thorough coagulation of the joint capsule. The buccal mucosa was closed, using interrupted fine black silk sutures, and 8 Gm. of sulfanilamide were placed in the neck wound which was then closed using interrupted fine black silk sutures. Two Penrose drains were placed at the extremities of the wound and a dry pressure dressing was applied.

Microscopic Description.—One node from the tail of the parotid, four from the upper end of the jugular, three from the lower end of the jugular and one buccinator node were all negative. Section through the center of the tumor showed well-differentiated epidermoid carcinoma. Section from the pterygoid fossa, including muscle and tumor, showed a small sliver of well-differentiated squamous carcinoma. The tumor was apparently completely excised.

All the nodes were negative. The tumor itself was well differentiated and, although large, was completely removed. The most unfavorable feature from the standpoint of prognosis was the extension of tumor into the loose tissue, for it is impossible to tell whether tumor has invaded the lymphatics and could be localized outside the point of excision (where it could remain dormant for variable periods of time).

Microscopic Diagnosis.—Lymph node, submaxillary: Epidermoid carcinoma, metastatic. The primary tumor arose questionably from the oral cavity, lip, lower. Lymph nodes, cervical: Hyperplasia 8/8 (Fig. 115).

The postoperative course was uneventful and the patient was discharged June 8 with a return appointment for two months.

Re-examination in August showed a mass measuring 2 by 2 cm. present at the posterior aspect of the horizontal incision immediately inferior to the angle of the mandible. There was no evidence of recurrent or persistent disease in the mouth.

An incision was made over the center of the mass and a biopsy taken which, by frozen section, proved to be positive. The tumor mass was outlined and the incision made 1 cm. around it. This incision was carried down perpendicularly to the underlying muscle of the pharynx and the second cervical transverse vertebral process. At this level the entire mass was undermined and removed en bloc. Hemostasis was obtained. The wound was packed open with sulfanilamide saline sponges. A stitch was placed through the tongue as a traction and the patient was returned to the ward in good condition.

Microscopic Description.—One section showed portions of the parotid salivary gland with some surrounding muscle and connective tissue. The capsule of the gland had been invaded by large masses of highly keratinized squamous tumor cells. Mitoses were infrequent.

Microscopic Diagnosis.—Soft tissue, submandibular region: Epidermoid carcinoma, Grade I. Postsurgical recurrence.

A tube flap was raised to provide more cover for the areas just superficial to the carotid bulb. The postoperative course was uneventful and the patient was discharged the last of September, 1942.

Examination on October 16 showed two superficial nodules, each measuring 1.5 cm., in the right midneck. There were also several small shotty supraclavicular nodes. In view of these two rapid recurrences it was suspected that the lesion was more malignant than the original pathologic diagnosis indicated and the only chance for cure was to re-excise these nodes.

Due to a series of colds, treatment was not instituted until November 6, at which time a right lower neck dissection was performed and a huge single split graft placed over the area (Fig. 116).



Fig. 114.



Fig. 115.



Fig. 116.

Fig. 114.—Photograph showing defect in left lower lip of lesion previously treated elsewhere and metastatic mass in right submaxillary area.

Fig. 115.—Photograph showing right neck and jaw following mandibular and upper neck dissection.

Fig. 116.—Photograph showing split thickness graft to defect in right neck and submaxillary area.

Microscopic Diagnosis.—Epidermoid carcinoma, ungraded. Postsurgical recurrence.

The split graft was 85 per cent successful. The infected 15 per cent was controlled with pinch grafts and the patient was discharged December 8. He was re-examined at two-month intervals until May, 1944, and at no time was there any evidence of local recurrence or metastatic disease in the opposite neck. Five chest films were taken in this seventeen-month period and all were negative for evidence of parenchymal disease.

In May, 1944, a leucoplakic indurated lesion was noted just medial to the right commissure of the upper lip. The patient attributed this to repeated trauma while shaving, but a biopsy was performed.

Microscopic Diagnosis.—Hyperkeratinization, hyperplasia, and chronic inflammation.

Examination in August showed the upper lip lesion to measure 1 cm. at the base. It infiltrated the lower lip just medial to the right lateral commissure for a distance of 0.5 cm. There was no evidence of recurrence in the right side nor metastasis to the opposite side. It was felt that the lip lesion probably represented carcinoma.

On August 17, under local anesthesia a v-excision of the upper lip was performed and a deformity of the left lower lip resulting from a previous endotherm excision elsewhere was corrected by a v-excision.

Microscopic Description.—The lesion was apparently completely excised.

Microscopic Diagnosis.—Epidermoid carcinoma, Grade I.

The postoperative course was not remarkable and the patient was discharged on August 23. At the last examination in February, 1945, there was no evidence of recurrent or metastatic disease.

Comment.—This case demonstrates the value of extensive surgical procedures even when metastases are apparently growing outside the capsule of a lymph node. Only one submaxillary node was involved but this was to be expected as the tumor was so well differentiated. Because of the size of the metastatic lesion, the surgical excision was of necessity rather close to the limits of the tumor. It was not unexpected, therefore, to have two local recurrences, one appearing in three months and the second five months after the original operation. It is worth while to re-excite such local recurrences in a well-differentiated carcinoma for they are likely to represent the only residual carcinoma present. In this instance, this supposition was substantiated by the clinical course for there has been no evidence of persistent disease for almost three years. These cases seldom develop recurrences after this time interval.

The occurrence of a new carcinoma of the upper lip was not unexpected, for a patient with one carcinoma of the lip has a much better chance of developing a second lesion than the patient of the same age developing a first carcinoma.

Case 38, J. R., EFSCH No. 4454

This 52-year-old white man was first seen in the clinic in August, 1942. There was a growth on the lower lip which had started as a small scab two years before. This lesion enlarged slightly and the local physician burned it with an electric needle. Following this, the area continued to form scabs which intermittently peeled off. The gradual enlargement prompted the patient to seek consultation elsewhere but for financial reasons no therapy was instituted at that time.

Examination revealed a somewhat exophytic, superficially ulcerated lesion measuring 3 by 3 by 1.5 cm. in the midline of the lower lip (Fig. 117). There were no palpable submental, submaxillary, or cervical nodes. A biopsy of the lip was done.

Microscopic Diagnosis.—Oral cavity, lip, lower: Epidermoid carcinoma, Grade I.



Fig. 117.



Fig. 118.



Fig. 119.

Fig. 117.—Photograph (full-face) showing large carcinoma of lower lip.

Fig. 118.—Photograph showing lower lip following radiotherapy.

Fig. 119.—Photograph showing metastatic mass in submental and left submaxillary space.

X-ray therapy totaling 3,450 r. to one 3 by 4 cm. field was given and the patient was discharged September 5. Re-examination in November showed the lesion well healed with only slight induration. Just to the right of the mid-line of the neck at the level of the hyoid bone there was a lymph node measuring 0.7 cm. which had grown rapidly during the course of a cold. A return appointment in one month was given with the idea of removing the node if still present.

Examination in December revealed a well-healed lip (Fig. 118) but the node now measured 1 cm. in diameter and was very hard. There was a second, firm node about the same size in the left submaxillary region. As the bulk of the lip lesion had been on the right side, this left submaxillary node probably represented a cross metastasis. A left complete and a right upper neck dissection in stages was planned, but the patient developed an acute cold and was allowed to return home with the explicit understanding that he would return in ten days.

This patient was not seen again until January 22, 1943. The first right submaxillary node now measured 2.5 cm. in diameter. The mass in the left submaxillary space now measured 7.5 cm. in diameter but was not fixed to the skin or bone (Fig. 119). An aspiration biopsy was performed on this node.

Microscopic Diagnosis.—Epidermoid carcinoma. There was a third mass measuring about 2.5 cm. in diameter in the submental area.

Roentgenograms showed no evidence of parenchymal disease.

It was felt that the tumor was no longer operable and the patient was referred to the Radiology Department. A total of 3,600 r. to each submaxillary region was given and the patient was discharged February 21 with an appointment to return in two months.

Examination in April, 1943, revealed a rounded definitely fluctuant and freely movable mass about 5 cm. in diameter in the left submaxillary area. Bilateral submaxillary edema was apparent but no masses were present on the right side. The patient was advised to enter the hospital for complementary treatment. A total of 3,000 r. was given to a small submaxillary field on each side.

The patient did not return as requested on July 7, nor did he ever come back in spite of repeated letters urging him to do so. The Social Service Department learned that this patient had entered another hospital on January 31, complaining of abdominal pains, nausea, loss of weight, and difficulty in swallowing. Roentgenogram examination there (according to reports) disclosed the presence of a large lobulated mass in the right mediastinum. The right lobe of the lung was hazy and contained a number of small, ill-defined densities, and the major portion of the lung showed confluent, poorly-defined shadows. The report read: "Findings were suggestive of a mediastinal tumor probably lymphoblastoma; however, massive metastatic carcinomatous lesions could also cause this picture." According to a letter, death occurred in that hospital on February 10. No autopsy was performed.

Comment.—This represents a rather infrequent sequence of events but serves to emphasize the importance of carefully timed appointments in the follow-up of patients with carcinoma of the lip. Unfortunately, in spite of the fact that this patient was informed of the seriousness of his condition and the great importance of his returning, he delayed until the disease got beyond the point of possible surgical control and died with probable metastases to the mediastinal lymph nodes and lungs.

Case 39, G. L., EFSCH No. 7014

An 83-year-old white man was first seen in the hospital in November, 1944, complaining of an unhealed lesion on the lower lip which had been present for approximately six years, occurring in the spot where a pipe was held. For the past two years it had increased in size and now measured 2 by 1 cm. A small mass under the right jaw had developed.

Examination showed two adjacent circular ulcerations on the vermilion border of the lower lip, each measuring approximately 1 cm. in diameter. They

were surrounded by areas of induration. In the right submaxillary region there was a node measuring 2 cm. in diameter attached to the horizontal ramus of the right mandible. There was diffuse surrounding induration.

On November 27 under local anesthesia a v-excision of the lower lip was performed.

Microscopic Diagnosis.—Oral cavity, lip, lower: Epidermoid carcinoma, Grade II. The lesion was apparently completely excised. An aspiration biopsy of the right submaxillary lymph node showed epidermoid carcinoma, Grade II, metastatic.

On December 11 under local anesthesia, an upper neck dissection with removal of a small portion of the right mandible was performed.

Microscopic Description.—Five submaxillary nodes were completely replaced by tumor and, in one, tumor was growing outside the capsule. Out of three nodes from the carotid bulb, one was partially replaced by poorly differentiated squamous carcinoma (Fig. 120). Because of the location of this node and the grade of carcinoma, disease was undoubtedly present in the lower lymph nodes. The prognosis was very poor.

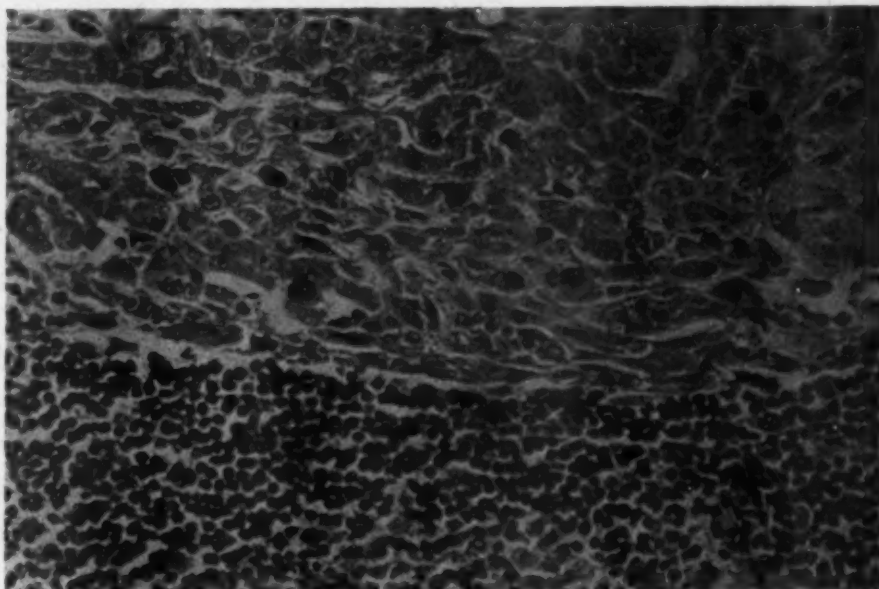


Fig. 120.—Photomicrograph (high-power). Relatively undifferentiated squamous carcinoma growing within a lymph node.

Microscopic Diagnosis.—Lymph nodes, submaxillary (right): Epidermoid carcinoma, Grade II, metastatic. Lymph nodes, cervical (carotid bulb): Epidermoid carcinoma, Grade II, metastatic.

On March 14, 1945, examination showed a contralateral metastatic node measuring 1.5 cm. in diameter in the left submaxillary fossa and another in the subdigastric region on the right. An aspiration biopsy was performed on the left submaxillary node.

Microscopic Diagnosis.—Epidermoid carcinoma, Grade II, metastatic.

On March 26, a left subtotal neck dissection was performed with removal of several small right cervical nodes.

Microscopic Diagnosis.—Lymph nodes, cervical (right): Epidermoid carcinoma, Grade II, metastatic (2/2). Lymph nodes, jugular-digastric (left): Hyperplasia (1/1). Lymph nodes, cervical (left): Epidermoid carcinoma, Grade II, metastatic (1/9).

The postoperative course was uneventful and the patient was discharged from the hospital on April 10. He died at home in June.

Comment.—This aged man had a small but undifferentiated carcinoma of the lower lip. If his general condition had permitted, a right radical neck

dissection would have been indicated because the tumor was an epidermoid carcinoma, Grade II. Five right submaxillary and one right carotid bulb nodes showed metastatic carcinoma. Carcinoma speedily developed on the opposite side, accompanied by evidence of disease in the lower right cervical lymph nodes. This sequence of events is unusual for a carcinoma of the lower lip, and any treatment except the most radical will be futile. It is particularly true also that if a metastatic node is present at the time the patient is first seen, then the chances of more than one node being involved is much greater than when the node appears several months after treatment of the primary lip lesion.

The authors are deeply indebted to the following men from the Ellis Fischel State Cancer Hospital for their contributions to this paper: Everett D. Sugarbaker, M.D., Medical Director and Chief Surgeon; David V. LeMone, M.D., Diagnostic Roentgenologist; Juan A. del Regato, M.D., Radiotherapist.

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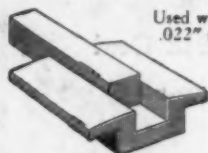
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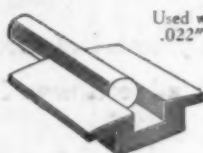
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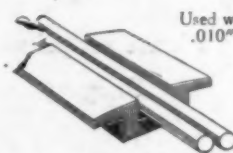
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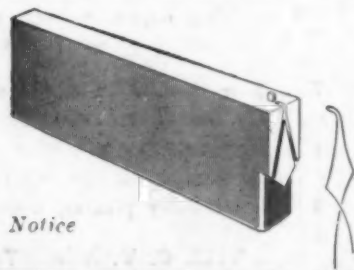
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Volume V

KURT H. THOMA, D.M.D.,* MARTIN WENIG, D.D.S.,† AND
SAMUEL I. KAPLAN, D.D.S.†

FUNCTIONAL DISTURBANCES FOLLOWING FRACTURE OF THE MANDIBULAR CONDYLE, AND THEIR TREATMENT

KURT H. THOMA, D.M.D.

FIVE cases have been seen during the past two years which presented major functional disturbances after fracture of the mandibular condyle. The following conditions were presented:

1. Nonunion causing crepitus and pain.
2. Pseudarthrosis causing partial ankylosis.
3. Synarthrosis due to dislocated condyle blocking movement of jaw.
4. Traumatic arthritis from malunion in angulated position of the fragments.
5. Partial ankylosis from malunion of a displaced or dislocated fractured condyle.

CASE REPORTS

The following case histories will be presented in order to discuss how complications following condylar fractures occur, how they are treated, and how they can be prevented:

Case 93

Nonunion of Unilateral Fracture of the Condyle

J. C., a 29-year-old married woman, was seen at my office because of pain and a cracking noise when moving the jaw.

The patient's past history was noncontributory. She had received a blow to the jaw five months before which was followed by a great deal of swelling; she had limitation of motion and could not open the jaw and was unable to masticate food. Roentgen examination at a suburban relief station was alleged to have shown no fracture or other injury to the mandible, and her local doctor said there was nothing seriously wrong with her. Her jaw remained immobilized by the swelling for weeks. She had received no treatment.

Examination revealed limited motion of the jaw; she was unable to open more than half the normal amount, and she could not move the jaw to the right. When the jaw was

*Oral Surgeon and Chief of Dental Department, Massachusetts General Hospital; Professor of Oral Surgery, Harvard School of Dental Medicine.

†House Officer, Dental Department, Massachusetts General Hospital.

thrust forward, it deviated to the left. Crepitus was heard and felt in the temporomandibular region on the left side. The region in front of the tragus of the ear was painful and tender to palpation.

Roentgen examination showed a fracture of the left condyle with lateral displacement of the neck of the condyle. There was no evidence of callus formation or bony union (Fig. 430).



Fig. 430.—X-ray of five-month-old subcondylar fracture with lateral displacement and nonunion.

The patient was advised that she had a fracture of the neck of the condyle, and that, because of the position of the fragment and the long elapse of time since the accident, an open reduction with freshening of the fracture surfaces and wiring fixation was necessary in order to get union with a good functional result.

Case 12, Previously Reported*

Partial Ankylosis Due to Pseudarthrosis Following Fracture Through Neck of Condyle and Treated by Condylectomy

P. M. (396690), a 19-year-old boy, was admitted on March 14, 1943, because of limitation of jaw motion and pain on mastication.

Ten years previously the patient had been struck by an automobile, suffering a fracture of the nose and right mandible. He was unconscious for twenty-four hours after the accident, and could not remember what type of treatment was used at the hospital to which he was taken. He had no subjective symptoms after discharge. In December, 1942, however, he noticed that his face appeared lopsided, and the left jaw felt tight when he opened his mouth. He had difficulty in chewing large mouthfuls.

Physical examination showed an apparently healthy youth in no pain. There was marked asymmetry of the face, with deviation of the facial bones to the right. The mouth could be opened barely a centimeter (Fig. 431). There was motion at the ramus in front of and below the tragus of the ear. Most of the natural teeth were in functional occlusion.

X-ray films taken in the anteroposterior position showed pseudarthrosis of the right ramus below the temporomandibular joint (Fig. 432). The temporal view showed normal temporomandibular articulation. In the lateral view an area of increased density was seen, indicating hyperostosis of the neck of the right condyle (Fig. 433).

The diagnosis was pseudarthrosis of the right temporomandibular joint.

*Clinic of the Massachusetts General Hospital, Vol. I, AM. J. ORTHODONTICS AND ORAL SURG. (Oral Surg. Sect.) 29: 550, 1943.



Fig. 431.—Pseudarthrosis of the right mandibular joint. Illustration shows the limited extent to which the patient could open the jaw before operation. (From Thoma and Kalil: AM. J. ORTHODONTICS AND ORAL SURG. [ORAL SURG. SECT.] October, 1943.)



Fig. 432.—X-ray showing pseudarthrosis of right mandibular joint.

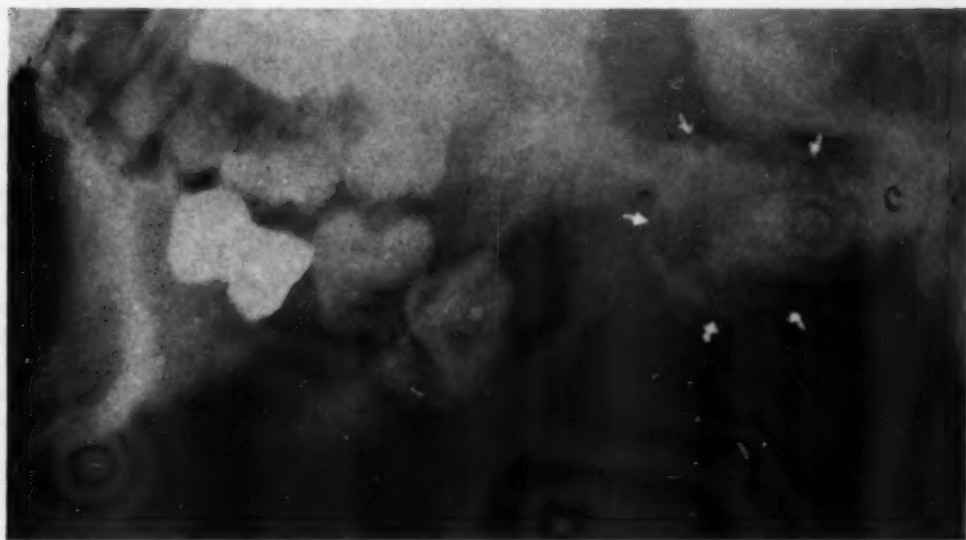


Fig. 433.—Lateral x-ray showing hyperostosis of right mandibular joint. Note condyle, C. (From Thoma and Kalil: AM. J. ORTHODONTICS AND ORAL SURG. [ORAL SURG. SECT.] October, 1943.)

The patient was admitted to the House and was treated by a mandibular condylectomy. He was discharged on the sixth postoperative day to be followed in the Outpatient Department. As a result of the operation and of exercises which were recommended, the patient was soon able to open his mouth to the width of three fingers (Fig. 434).

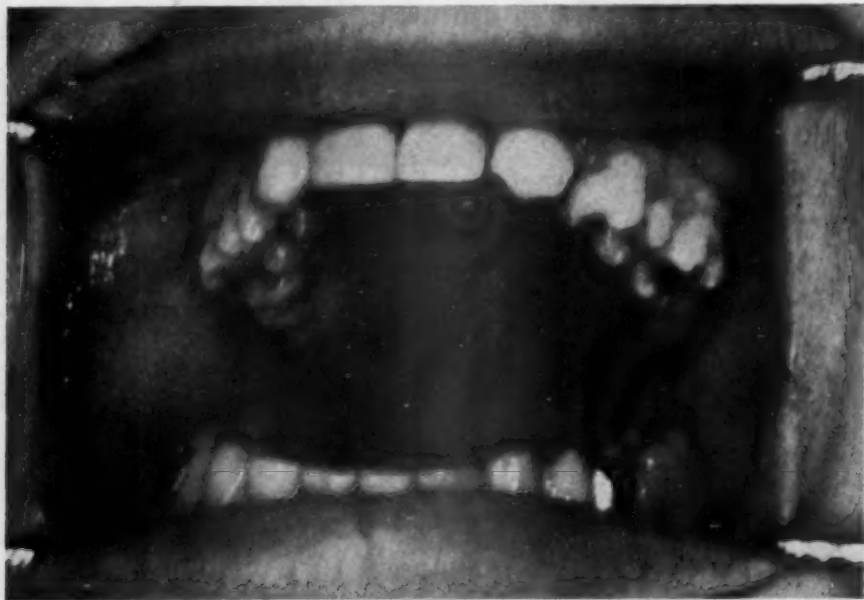


Fig. 434.—Photograph showing the extent to which the jaw could be opened three days after operation. (From Thoma and Kalil: *AM. J. ORTHODONTICS AND ORAL SURG.* [ORAL SURG. SECT.] October, 1943.)

Case 94

Synarthrosis Due to Fracture Dislocation and Attachment of Condyle in Displaced Position in the Case of Bilateral Condylar Fractures

M. B. (489462), a 36-year-old married woman, a truck driver, entered the hospital on May 11, 1945. She was unable to open her jaw because of bilateral fractures of the condyles.

On Feb. 2, 1945, the patient was injured in an accident in which her truck collided with a snowplow. She remembers little about the accident, and was treated by a physician for a slight cut over the lower jaw. Because of pain and a loose maxillary tooth, she sought the services of her dentist who suspected multiple fractures and referred her to an oral surgeon. He found that she had a fracture of the right maxilla and fractures of both condyles, with dislocation. He performed a closed reduction and immobilized her jaw with intermaxillary wiring. When the wires were removed after eight weeks, the patient could not open the jaws. A second closed reduction was performed because of postoperative displacement of the condyles. This was temporarily successful, but the right condyle became further displaced causing pain and preventing normal mobility of the jaw. The patient was referred for an open reduction of the fracture.

Examination showed the maxillary fracture to be well healed. She was unable to open her jaw. Attempted movement caused pain on the right, but none on the left. Palpation of the region of the right mandibular joint caused pain. Otherwise the physical examination was negative.

X-ray examination showed fracture of both sides of the mandible. The condyle on the left was medially displaced and apparently caused no interference, while the right condyle was displaced laterally with the fractured end close to the skin and far from the fracture surface of the ramus. It appeared to be wedged between the posterior border of the ramus and the tympanic plate (Fig. 435). In comparing this x-ray with previous ones, it was quite evident that the condyle had become considerably displaced since the first examination.

Open reduction of the right condylar fracture with wiring fixation was advised, and if this did not give a satisfactory result, the left side would be operated on later.

The operation was performed on May 12 under intratracheal gas, oxygen, and ether anesthesia. After the usual preparation of the temporal and preauricular region, an angu-

lated vertical incision was made in front of the ear, extending as far as the attachment of the lobe to the skin. The subcutaneous tissue was divided and the superficial orbital artery cut and tied. The zygomatic arch was then dissected down upon and demonstrated, and the glenoid fossa opened by incising the external part of the capsule. The ramus could be seen deep in the wound when the jaw was moved, but it was very difficult to find the condyle, as it was dislocated and at a considerable distance down on the medial surface of the ramus,

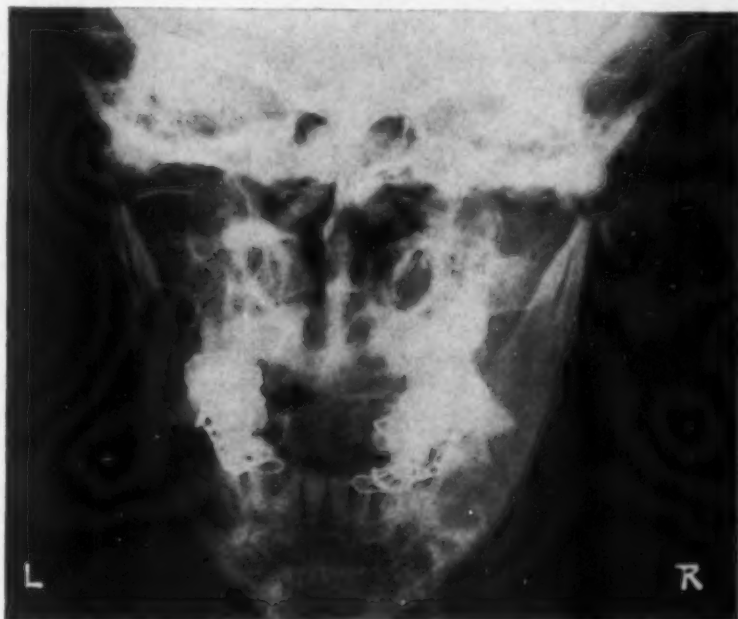


Fig. 435.—Anteroposterior x-ray showing fracture of both condyles. On the left there is a fracture dislocation with medial displacement; on the right the lateral displacement of the condyle caused synarthrosis.

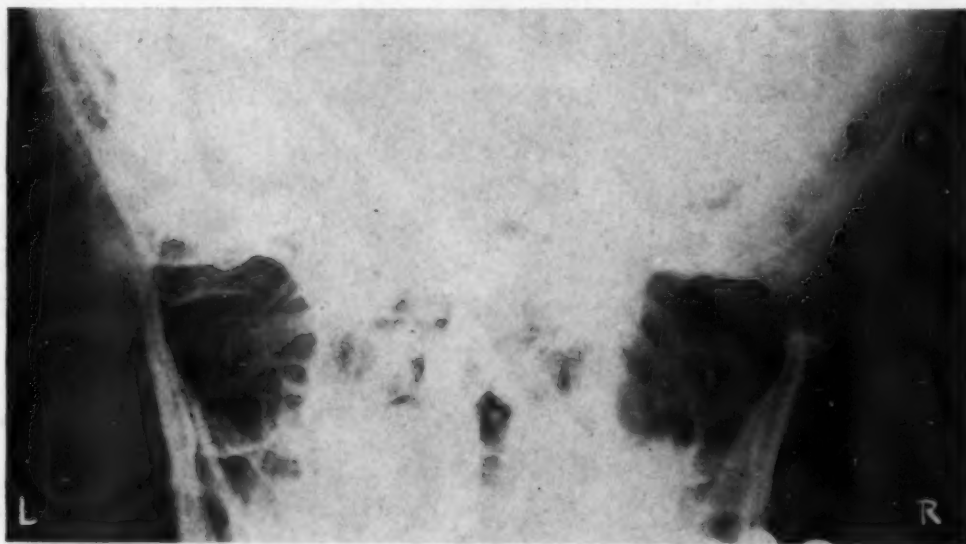


Fig. 436.—Postoperative x-ray showing reduction of the fracture on the right and fixation by interosseous wiring.

and was wedged between the ramus and the mastoid process. By means of sharp hooks it was elevated into the glenoid fossa. A hole was drilled through its neck, after which a 25 gauge stainless steel wire was inserted through the hole. The fractured end of the ramus was then pushed into the wound by pressing at the angle of the jaw. The edge of the wound was retracted, and the ramus was exposed sufficiently to drill another hole. With a stainless steel loop, the first wire was drawn into this hole. The fracture was reduced and fixed by twisting the wire, and the end was cut and turned down on the bone. In order to prevent dis-

location of the head of the condyle, a chromatinized catgut, No. 00, was drawn into the periosteum on the outer side and attached to the temporal fascia above the zygomatic arch. The subcutaneous tissue was closed with interrupted catgut sutures, and the skin with subcuticular sutures. A rubber-dam drain was inserted into the wound at the site of the fracture in order to prevent a hematoma from forming. The eye reflex was tested and found to be normal. Immediately after the operation the jaw could be moved freely without obstruction, and it was felt that the deformity on the left would not interfere greatly with a good functional result. At least, healing of the condyle in the dislocated position should not give the patient more debility than would result from excision of the condyle. The mandible was immobilized by wiring together the Jelenko splints which had been attached to the jaws previously. The patient was discharged from the operating room in good condition.

The postoperative course was uneventful except for some pain in the chest. The temperature was 99.2° F., and penicillin was injected intramuscularly, 12,000 units every three hours. This was discontinued after the fifth day when danger from pneumonia had passed. The patient had the usual swelling over the area operated on, and slight ecchymosis because the drain which was inserted to prevent a hematoma came out of the wound while the patient was recovering from the anesthesia. On May 16 she felt very well and was allowed to walk around the hospital; on May 21 she was discharged in the care of the oral surgeon who referred her to me. On discharge her mandible was well immobilized; the suture had been removed and the wound had healed by first intention. There was no Bell's sign, evidence that the facial nerve supplying the eyelid had not been injured. Postoperative x-ray examination showed that the fracture was well reduced and fixed by interosseous wiring (Fig. 436).

Case 95

Partial Ankylosis Following Fracture of the Mandibular Condyle, Treated by Osteoarthrotomy

B. K. (478523), a 25-year-old woman, was admitted to the hospital on Feb. 2, 1945, with ankylosis of the right mandibular joint. The patient had been involved in a coasting accident many years before, and an automobile accident about four years before, either of which might have caused a fracture of the right condyle resulting in malunion. She was suffering considerable pain at the site of the right mandibular joint which became severe when she tried to open the jaw. The pain caused by forcing the jaw open extended over the top of her head, and was referred to the ear. The pain was often severe enough to confine her to the house.

Examination showed a thin, nervous woman with tenderness over the right mandible near the joint. She could open the mouth only about $\frac{3}{4}$ inch; she could not move the jaw to the left, but motion to the right was normal. There was a history of tuberculosis, but there were no physical signs present except for a thickened pleura at the right base posterior.



Fig. 437.—Anteroposterior x-ray showing malunited fracture through the neck of the condyle with angulation.

X-ray examination showed malunion of the right mandibular condyle with angulation of the fragments (Fig. 437). Marked increased density was noted in the lateral view, probably due to new bone formation causing ankylosis of the jaw (Fig. 438).

Diagnosis: Malunion of condylar fracture and partial ankylosis.

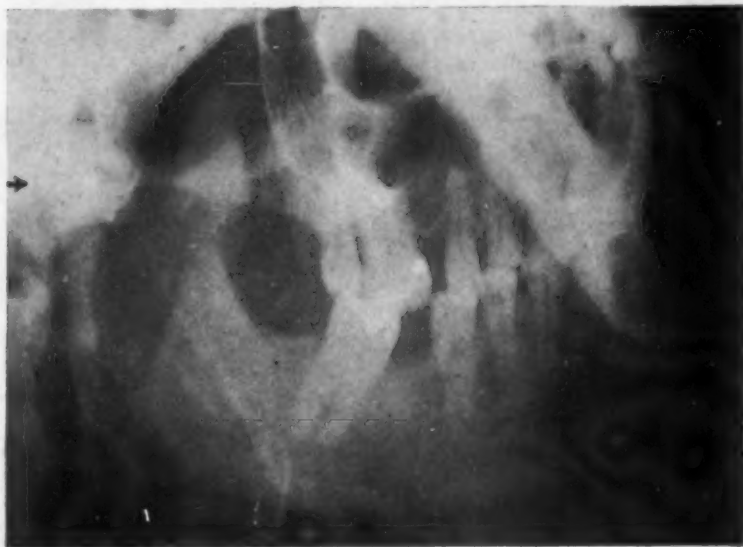


Fig. 438.—Lateral view showing increased density and bone formation causing ankylosis.

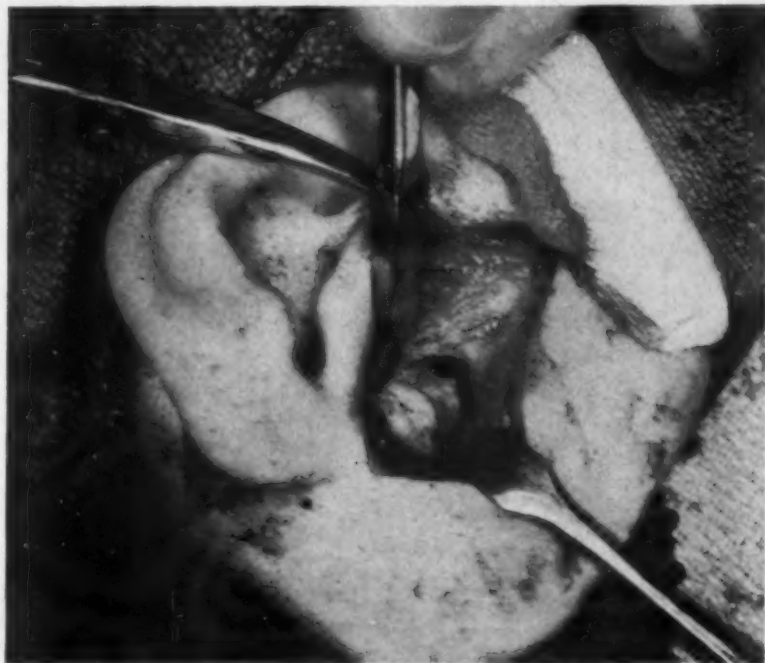


Fig. 439.—The articulating surface of the condyle, facing in an anterior direction, is exposed.

On February 3, under endotracheal gas, oxygen, and ether anesthesia, an osteoarthrotomy was performed. After the usual preparation of the skin, an angulated vertical incision was made in front of the right ear. The subcutaneous tissue was divided and the superficial orbital artery and vein were tied and cut. The zygomatic arch was then dissected down upon and its inferior border demonstrated. The site of the ankylosed joint was thus located. Investigation showed that there was very little motion in the joint when the anesthetist moved the jaw up and down. The joint capsule was opened and the condyle was found deformed, with its articulating surface facing in an anterior direction (Fig. 439), the meniscus being jammed between it and the eminentia articularis. The meniscus was removed by detaching

it from the capsule (Fig. 440). Osteotomy was performed at the condylar neck by means of burs and osteotomes. Then an osteotome was inserted into the joint area and the condyle broken away from the glenoid fossa. The external pterygoid muscle was detached and the head of the condyle removed. The amputated ramus was then made smooth. There was considerable bone bleeding which was arrested by packing fibrin foam soaked in thrombin over the cut surface. The subcutaneous tissue was then closed with catgut sutures, and the skin by a subcuticular suture (Fig. 441). A rubber-dam drain had been previously inserted to prevent a hematoma from forming, and a dry dressing was applied with an elastic bandage.



Fig. 440.—Removal of the meniscus.

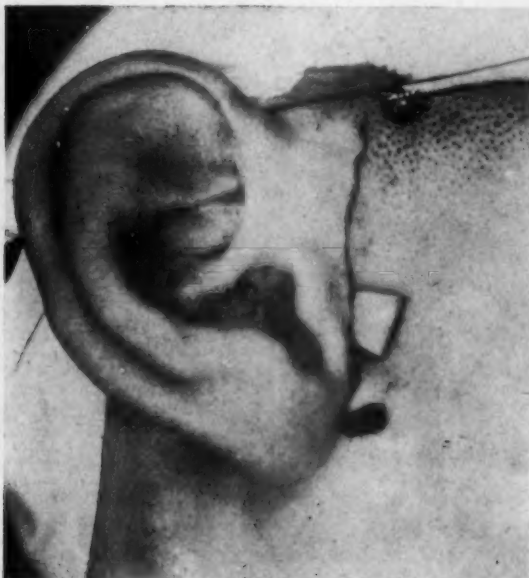


Fig. 441.—Incision closed by subcuticular suture.

Sulfadiazine, 6 Gm., was given on the day of entry and continued for four days post-operatively. There was some oozing of blood from the wound; about $1\frac{1}{2}$ c.c. were lost. The rubber-dam drain was removed the day following the operation, and the area cleaned with boric alcohol. A dry dressing and a Barton bandage were applied. The patient complained of noises and pain in the right ear, and on February 6 a cotton plug was found and removed

from the ear. Apparently the cotton inserted into the external meatus to prevent blood from entering had slipped out of sight during the operation. The following day the ear was normal.

X-ray examination on February 6 showed the right condyle had been resected. The joint space was wide and free. An old sclerosis of the condylar fossa was noted.

The patient was discharged on February 10 to be followed at the office.

When seen on February 19, the patient stated that she was greatly benefited by the operation. She had good motion of the jaw, and volunteered the fact that for the first time she was able to masticate food on her posterior teeth.

Case 96

Partial Ankylosis Following Fracture Dislocation of the Mandibular Condyle Treated by Osteoarthrotomy

M. M. (477386), a 30-year-old woman entered the hospital on Jan. 22, 1945, with a complaint of inability to open the jaws.

Seven years before the patient had been involved in an automobile accident and had sustained a fracture of the right condyle. This was reduced and fixed with intermaxillary

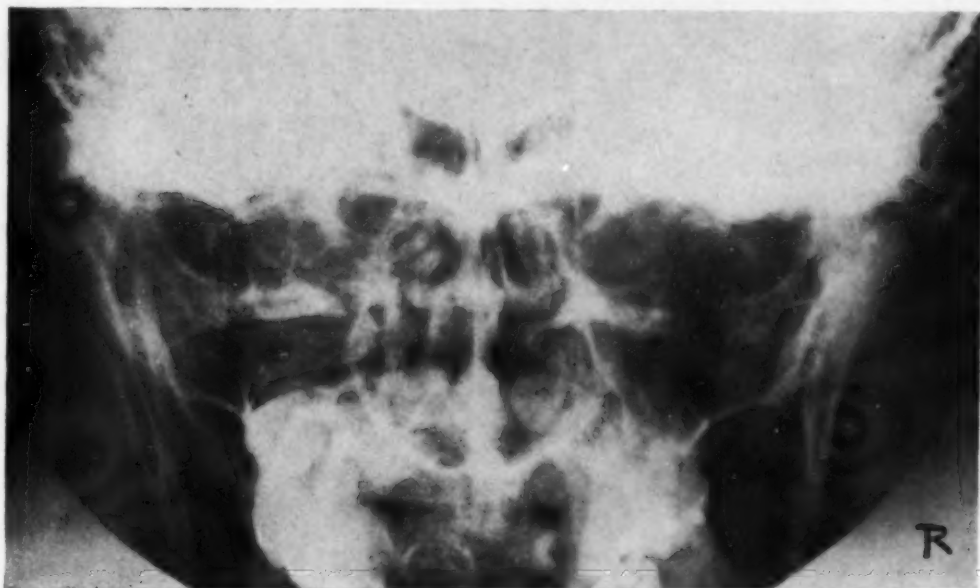


Fig. 442.—Old condylar fracture showing the right condyle united in a displaced position.



Fig. 443.—Lateral view showing new bone formation about the old fracture causing ankylosis.

ligation and rubber bands, which she wore for six weeks. After this treatment she had pain when eating or talking for a long period. This improved, but she was unable to open the mouth more than $\frac{1}{8}$ inch.

Examination showed inability to open the mouth more than $\frac{1}{8}$ inch anteriorly, and limited motion of the jaws on lateral excursion. Sliding of the right condyle was not demonstrable on palpation during mandibular movements. There were several teeth missing in the upper jaw including the incisors.



Fig. 444.—Incision to expose zygomatic arch.

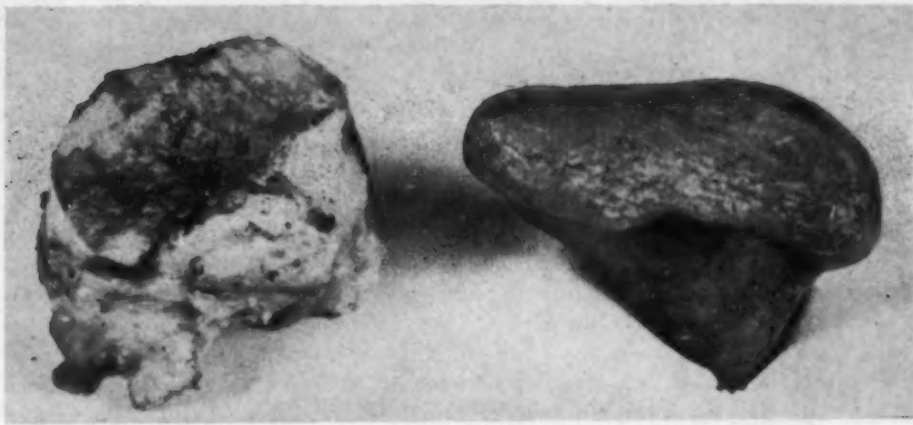


Fig. 445.—Photograph after condyle was excised.

X-ray examination showed the right condyle to be displaced medially (Fig. 442). There was extensive new bone formation about the old fracture (Fig. 443), which probably was a fracture dislocation.

Diagnosis: Ankylosis of the temporomandibular joint.

On January 23, under endotracheal gas, oxygen, and ether anesthesia, an osteoarthrotomy was performed. After the usual preparation of the skin an angulated incision was made in front of the right ear. The subcutaneous tissue was divided and the superficial orbital artery



A.

B.

Fig. 446.—Photograph of excised condyle (A) showing compound articular surface due to old fracture; B, normal condyle for comparison.



Fig. 447.—Photograph taken on tenth postoperative day after removing suture.

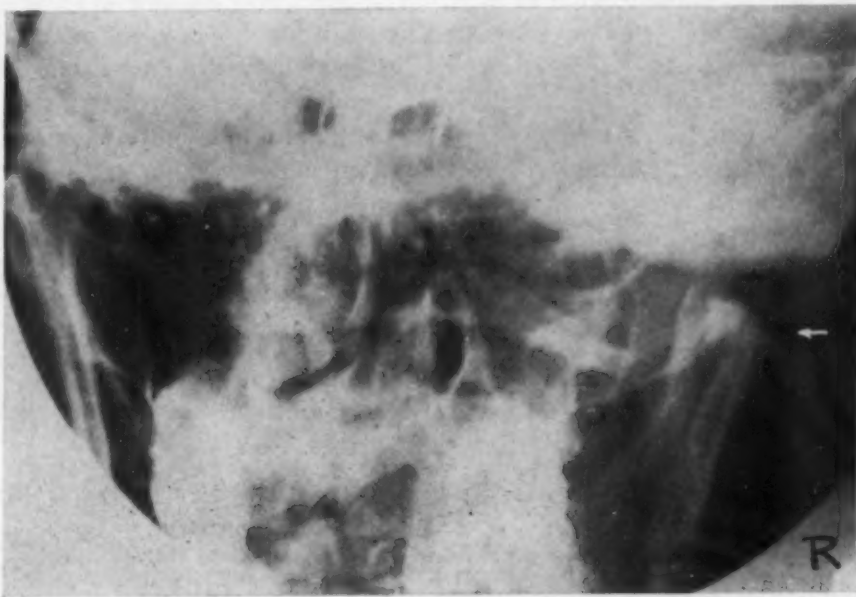


Fig. 448.—Postoperative x-ray after excision of condyle.

and vein were cut and tied. The zygomatic arch was then dissected down upon and its inferior border demonstrated (Fig. 444). The site of the ankylosed joint was thus located. Investigation showed that there was very little motion in it as the anesthetist moved the jaw up and down. Osteotomy was performed at the neck by means of burs and osteotomes. Then the osteotome was inserted into the joint area and the condyle broken away from the glenoid fossa. The external pterygoid muscle was detached and the head of the condyle removed (Fig. 445). Examination of the condyle showed that there was great deformity; several pieces of bone projected away from it, and it presented a compound flat articular surface which probably was the result of malunion of the fracture (Fig. 446). The amputated ramus was then made smooth. There was considerable bone bleeding, and this was arrested by packing fibrin foam soaked with thrombin over the cut surface. The subcutaneous tissue was closed by means of catgut sutures, and the skin by a subcuticular suture. A rubber-dam drain had been previously inserted to prevent the formation of a hematoma, and a dry dressing was applied with an elastic bandage.

An intravenous infusion of 1,500 c.c. dextrose was given postoperatively. Sulfadiazine, 3 Gm., on the day of operation, and 6 Gm. on each of the next four postoperative days, was administered. On the first postoperative day the drain was removed and on the tenth day the sutures (Fig. 447).

X-rays on January 24 showed that the right condyle, together with the proliferative bony mass, had been resected (Fig. 448). The patient made an uneventful recovery and was discharged on January 31. She was able to open her jaw without obstruction and to masticate food without pain.

DISCUSSION

A study of these five cases allows us to draw the following conclusions:

1. Inadequate immobilization tends to nonunion and pseudarthrosis associated with limitation of motion and pain. In spite of the abundant callus which is formed in condylar fractures, adequate immobilization of the mandible is absolutely essential in order to get union. Cases 93 and 12 disprove the statements made by Zemsky (1926),* Guy (1928),† and Steadman (1939)‡ that immobilization is unnecessary and even inadvisable. They advocated active movement of the jaw, and Dufourmental (1929)§ controlled function by means of intermaxillary elastics. In Case 93, position of the fragments was not unfavorable for obtaining a good result. In Case 97, a subcondylar fracture (Fig. 453) in which the position was not unlike that shown in Fig. 430, the extensive callus thrown out caused union in a period of five weeks, during which time the jaw was immobilized. In Case 93, there was no callus visible in the x-ray after a period of twenty weeks. Another important factor in causing nonunion is tissue which may become interposed. A diastema between the fragments is not a great hindrance; it will be bridged over by callus formation in this region, but, if muscle is interposed, healing will be prevented and nonunion will result. In addition, nonunion may occur if the fragment is displaced to the medial or lateral side with the fracture surfaces away from one another. In such cases the fractured surface of the ramus is in contact with the smooth surface of the condylar neck (Fig. 435).

Case 12, in particular, proves that Walker's|| (1942) statement is correct, namely, that active movement or movement controlled by elastics leads to the formation of a false joint, because after ten years there was definite evidence of pseudarthrosis.

*Zemsky, J. L.: New Conservative Treatment Versus Surgical Operation for Displaced Fractures at Neck of Mandibular Condyle, *D. Cosmos* 68: 43, 1926.

†Guy, W.: Injuries of Jaws, *Brit. D. J.* 40: 904, 1928.

‡Steadman, S. J.: Symposium on Fractures of Jaws, *Brit. D. J.* 67: 273, 1939.

§Dufourmental, L.: *Chirurgie de l'articulation temporomaxillaire*, Paris, 1929, Masson et Cie.

||Walker, D. G.: Fractures of Ramus, Condylar and Coronoid Processes of Mandible, *Brit. D. J.* 72: 265, 293, 1942.

2. Displacement of a dislocated condyle may cause synarthrosis. By muscle pull the condyle may be drawn away from its original location, and it may attach itself to the jaw by callus formation. The elevator muscles which draw the mandible up may mechanically block mandibular movements in certain cases. In Case 94 this condition was demonstrated at the time of operation and corrected by condylectomy.

3. Malunion in grossly displaced position may cause traumatic arthritis or deformity of the condyle leading to partial or complete ankylosis of the jaw. Angulation of the fragments (as shown in Fig. 437) causes functional disturbances in the joint with pain and limitation of motion. Intra-articular hemorrhage or comminution of the head of the condyle, or a fracture close to the capsule may produce a callus from which a dense mass of bone may form around the condyle, interfering with the motion of the jaw. This condition was demonstrated in Cases 95 and 96, and is best seen in a lateral view (Fig. 443).

4. Malunion in fracture dislocation is serious in proportion to the age of the patient. In young persons some sort of a joint will develop with fairly good function resulting. In older patients limited motion and partial ankylosis is more likely. Case 96 is a typical illustration.

TREATMENT OF COMPLICATIONS CAUSING DYSFUNCTION

Nonunion.—The treatment of this complication depends, first of all, on the duration of the condition. Early nonunion in favorable cases may be treated simply by immobilization of the mandible, especially if the original immobilization was inadequate. Older cases may require freshening of the fracture surfaces, the removal of interposed tissue, and internal wiring fixation, which should not be withheld since an open operation is required in such cases.

Pseudarthrosis.—In pseudarthrosis condylectomy gives satisfactory results. It increases the mobility of the mandible and eliminates painful sensations. There is little to be gained by attempting to achieve union between the fragments as there is always loss of bone between them, and freshening of the fracture would aggravate the situation. It would not be possible to establish contact without removing teeth to draw the ramus up or incise the capsule to draw the condyle down.

Traumatic Arthritis.—In most cases there are gross changes in the articulation which are beyond repair. Even though an osteotomy may be performed to eliminate angulation, the irregularities of the articular surfaces and the deformity of the meniscus cannot be eliminated; the results of an anatomic alignment of the bone, therefore, would give no satisfaction.

Synarthrosis.—If this condition is due to interference with motion by an interposed dislocated condyle that may be attached or unattached, it can be remedied by an open operation and internal fixation. In cases in which the condyle has undergone resorptive changes, or old cases where the condyle has been comminuted, excision of the fragment or fragments is the only possible procedure.

Ankylosis.—In cases in which partial or complete interference with function is due to changes in the articulation, especially those in which there is considerable new formation of bone, osteoarthrotomy is indicated.

Malocclusion.—In most of these cases malocclusion is present in one form or another. Open-bite is quite common, and deviation of the teeth to one side or the other, in addition to a forward or backward shifting of the mandible. All these disturbances, although of importance, are secondary in nature and are

generally eliminated by correcting the deformity of the jaw. In many cases, however, judicious grinding of the teeth, the changing of an artificial denture, or bridges may aid in establishing a new resting point for the teeth. In other cases, especially those in which a condylectomy or arthroplasty has been performed, the use of intermaxillary elastics for a short period of time will help the patient to achieve proper articulation and balance during mastication.



Fig. 449.—Vertical exposure shows the articular surfaces of the condyles.

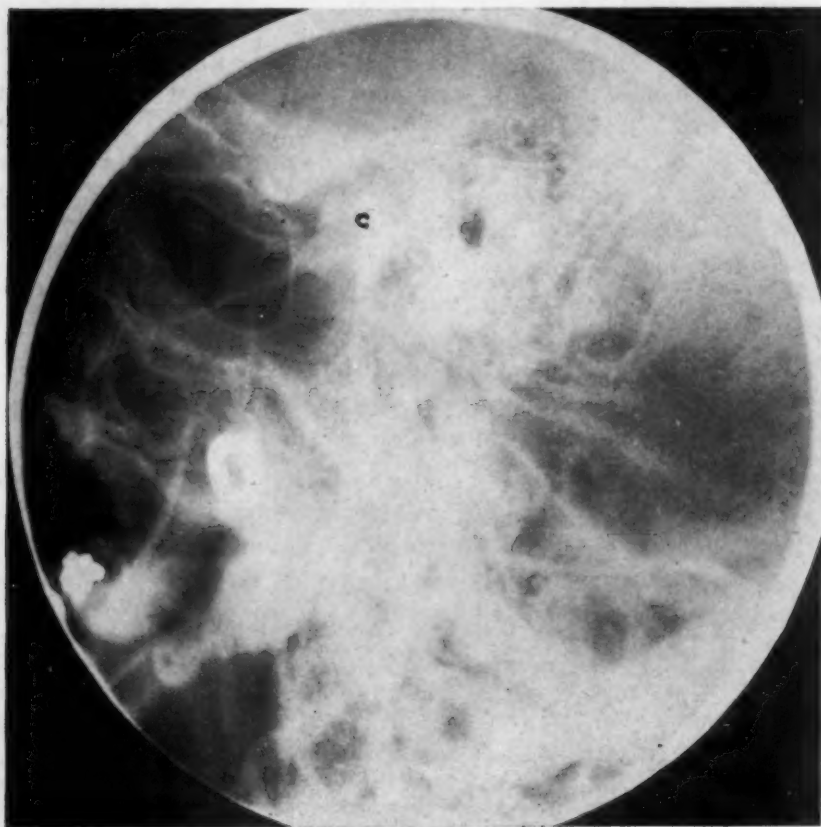


Fig. 450.—Temporomandibular exposure. The joint space is obliterated by bone which caused ankylosis of the condyle (C). This was taken in an open mouth position, showing that the condyle remained stationary.

PREVENTION OF COMPLICATIONS IN CONDYLAR FRACTURES

The prevention of many of these complications is possible, first by making a careful diagnosis of the type of fracture presented by the patient, and second, by applying the proper type of treatment.

Clinical examination should include palpation of the condyle by placing a finger over the condylar area and into the orifice of the external meatus of the ear, the elicitation of crepitus by means of a stethoscope, examination of the mobility of the jaw in forward motion, lateral excursion, and hinge motion, and a careful study of the occlusion.

X-ray Examination.—The development of new positions from which condylar fractures may be investigated has aided greatly in the correct diagnosis of complications. The lateral film without overlapping of the cervical vertebrae (Fig. 455), and an anteroposterior view (Fig. 430) taken at an angle of 10 degrees are the minimum in an x-ray examination. The vertical exposure often gives a fine view of the articular surfaces of the condyles (Fig. 449), while the temporomandibular exposure is useful for details of interarticular conditions (Fig. 450). It shows the outline of the condyle and glenoid fossa. In case of ankylosis and hyperostosis it is of little help as it shows nothing but an indefinite area of calcification which obliterates the limited landmarks generally presented for inspection and diagnosis.

Selection of Operative Procedure.—Simple fractures through the condylar neck may be treated conservatively with the expectation of a very excellent result; even in cases in which there is some displacement and space between the fragments, as in Case 97, healing will take place because of the great amount of callus formed in this region.

Case 97

Left Subcondylar Fracture With Fracture of the Left Horizontal Ramus of the Mandible

A. C. (482475), a 28-year-old man, entered the hospital on March 17, 1945, with pain in the left condylar region and inability to occlude the teeth.

The afternoon of admission the patient was struck on the jaw by a flying jack. Examination showed a compound fracture of the mandible through the distal portion of the right lateral incisor. The right posterior fragment was displaced slightly upward by the elevator muscles and the anterior fragment downward by the depressor muscles. There was some bleeding from the compounded area.

The x-ray examination revealed fractures through the left side of the mandible just below the condyle with some overriding and outward rotation of the proximal fragment, shown in the anteroposterior view (Fig. 451) and forward displacement in the lateral view (Fig. 452). A fracture through the right side of the mandible just below the canine and incisor teeth was shown in an additional film.

On March 19 reduction of the fractures and extraction of one tooth were performed. Under intravenous pentothal sodium anesthesia, after the usual preparation of the face and mouth, Jelenko splints were attached to both upper and lower dental arches. The right lower canine was extracted, because it was in the fracture line. Then the second fracture was reduced as accurately as possible, and the mandible immobilized with vertical and oblique elastics. This brought the teeth into good occlusion, and therefore would probably take care of the condylar fracture on the left as well as the mandibular fracture on the right canine region. The reduction was to be checked by x-ray, and if the reduction of the condylar fracture was not satisfactory, an open reduction was to be done. X-ray examination on March 22 showed the fragments about the anterior fracture held in good position and alignment, but the fragment of the left condyle was still overriding. There was some lateral displacement of the condylar portion.

After the operation the patient received an intravenous infusion of 1,500 c.c. of 5 per cent dextrose in water, and sulfadiazine, 3 Gm. on the first day and 6 Gm. on the second and third days. On the first postoperative day he was given 32 units penicillin and 128,000 units intramuscularly on the next six days. He made an uneventful recovery and was discharged on March 28 to be seen at the office.

The patient was seen several times and the mouth was sprayed and the intermaxillary fixation checked. On April 23 the mandible was mobilized after the x-ray showed good callus formation in spite of the distraction of the fragments (Fig. 453). He was again seen on April 30; he stated that his jaw was functioning normally, and that he was able to masticate hard food without difficulty.

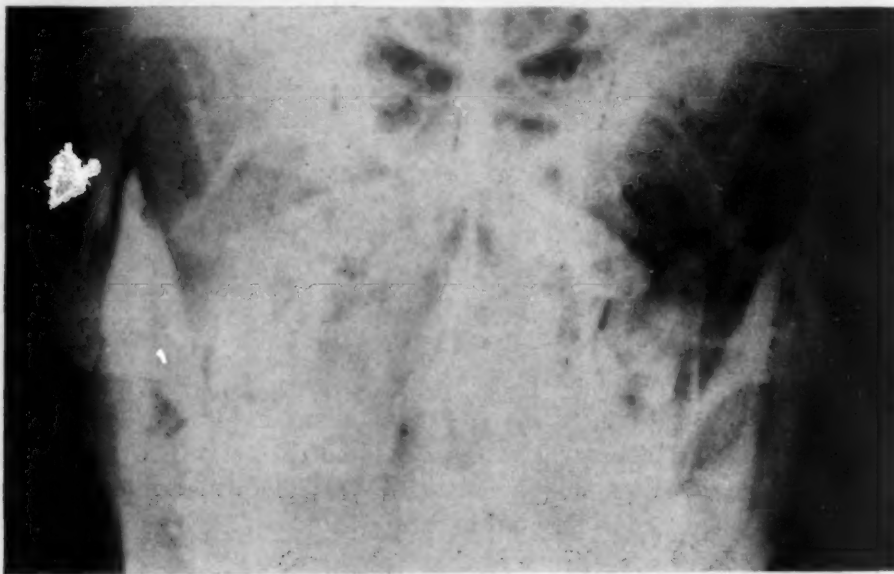


Fig. 451.—Anteroposterior view showing subcondylar fracture with lateral displacement.



Fig. 452.—Lateral view (touched up) showing condylar fracture with forward displacement and overriding.

Subcondylar fractures are less likely to cause functional abnormalities than condylar fractures. In complicated condylar fractures, better than average results can be achieved by open reduction and direct fixation. Open reduction gives an opportunity to correct displacement by muscle pull, which can never be done

by closed manipulation; it permits the elimination of a diastema between the fragments caused by an interposed small piece of bone or soft tissue, and allows proper impaction of the fracture. In fracture dislocation, the head of the condyle may be properly positioned in the glenoid fossa and its relation to the meniscus adjusted. The capsule may be repaired by suturing.

Open reduction is also advantageous because it gives opportunity for fixation of the fragment under direct vision.



Fig. 453.—X-ray taken five weeks after immobilization, showing callus formation (arrow) between the two fragments.

In fractures in which the condyle is not dislocated, interosseous wiring gives excellent results. The method has been described elsewhere in detail.* It prevents postoperative displacement which may occur while intermaxillary fixation is applied for the immobilization of the mandible. Although this danger may be lessened by applying Jelenko splints before the operation and placing a small number of elastics to hold the jaw in occlusion, permitting manipulation of the jaw under the drapes during the operation. In cases in which complete immobilization is not possible because of the condition of the teeth, internal wiring fixation is particularly useful.

In fracture dislocation it is often necessary to use fixation to hold the condyle in position after it is replaced into its anatomic position. Two methods are recommended and their use is described in the two following case reports. In one the fracture was immobilized by means of internal wiring fixation and skeletal fixation. The latter prevents redislocation during and immediately after the operation. It may be removed on the fifth or sixth postoperative day. The other case shows the use of a Sherman plate which both immobilized the fracture and held the condyle in place. Therefore, one appliance takes the place of two. Whether plates are as satisfactory here as wires cannot be ascertained as yet. Skeletal fixation is easily removed. Stainless steel wires, we know, do no harm, and may remain permanently.

*Thoma, K. H.: Fractures and Fracture Dislocations of the Mandibular Condyle: A Method for Open Reduction and Internal Wiring and One for Skeletal Fixation, With a Report of Thirty-Two Cases, *J. Oral Surg.* 3: 3, 1945.

Case 98

Open Reduction of Mandibular Fracture Dislocation With Skeletal and Internal Wiring Fixation

D. D., a 26-year-old housewife, was seen because of a condylar fracture which prevented opening of the jaw and moving it forward.

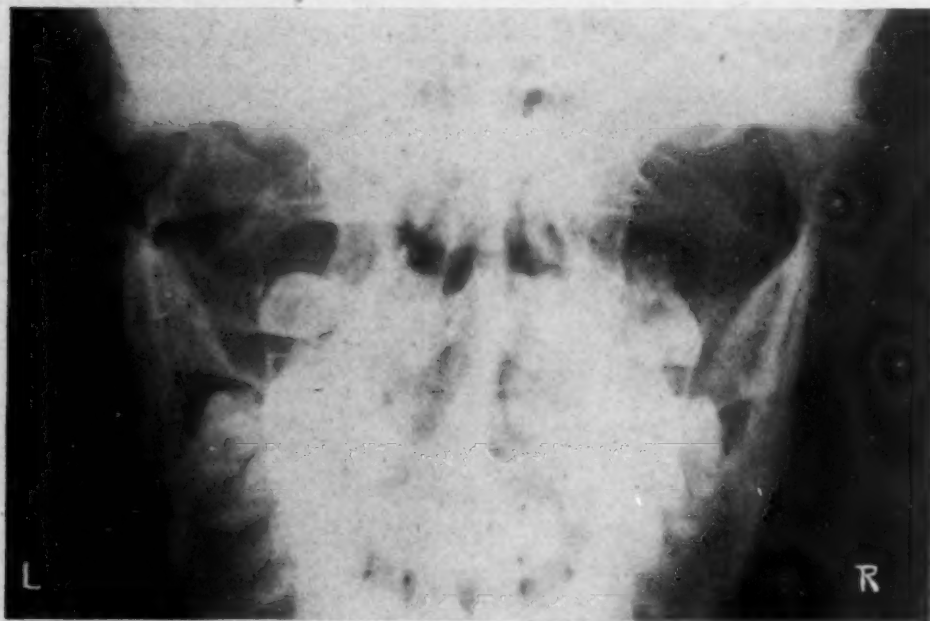


Fig. 454.—Anteroposterior view showing fracture dislocation on the left with medial displacement, and an old fracture on the right with the condyle in a good anatomic position.

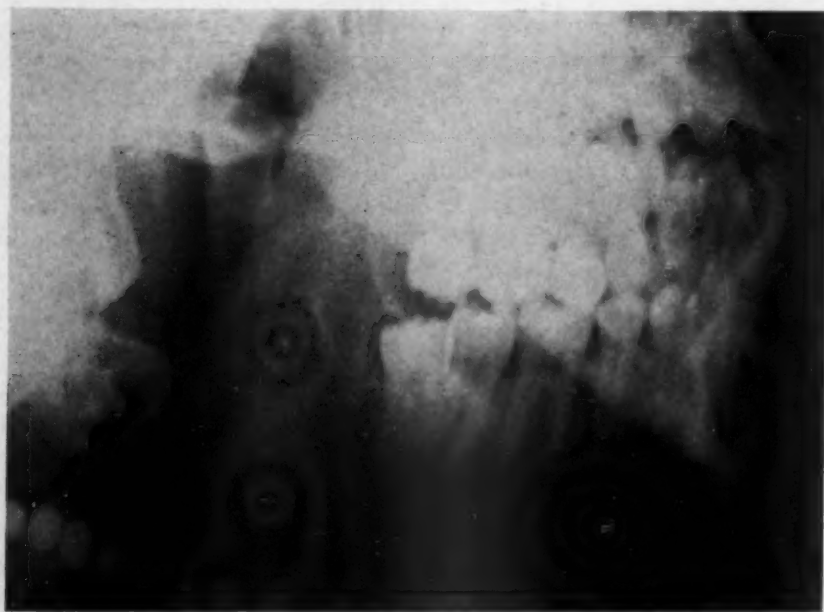


Fig. 455.—Lateral view of left side showing slight rotation of the condylar fragment.

The patient had been in an automobile accident when a baby and had injured her nose. In 1939 she sustained injuries when she fell downstairs. She had a cut on the chin which was closed by suture. The mandible was thought to be dislocated and a Barton bandage was applied for two and one-half weeks by an oral surgeon. When the bandage was removed she could not move her jaw forward. The patient consulted me and I found bilateral fractures

through the condylar necks. The patient was treated by intermaxillary fixation; there was very little displacement and a good result was obtained. After discharge the patient was asymptomatic until she was involved in an automobile accident three weeks before admission, sustaining lacerations of the left knee and left jaw.

Examination showed inability to open the jaw normally or to move it to the right. She had some pain in the region of the left mandibular joint, especially when talking or eating.

X-ray examination showed a fracture dislocation of the left condyle, which was displaced medially (Fig. 454). The condyle on the right, fractured six years ago, was anatomically normal, and in good position. The lateral view showed slight rotation of the condylar fragment (Fig. 455).

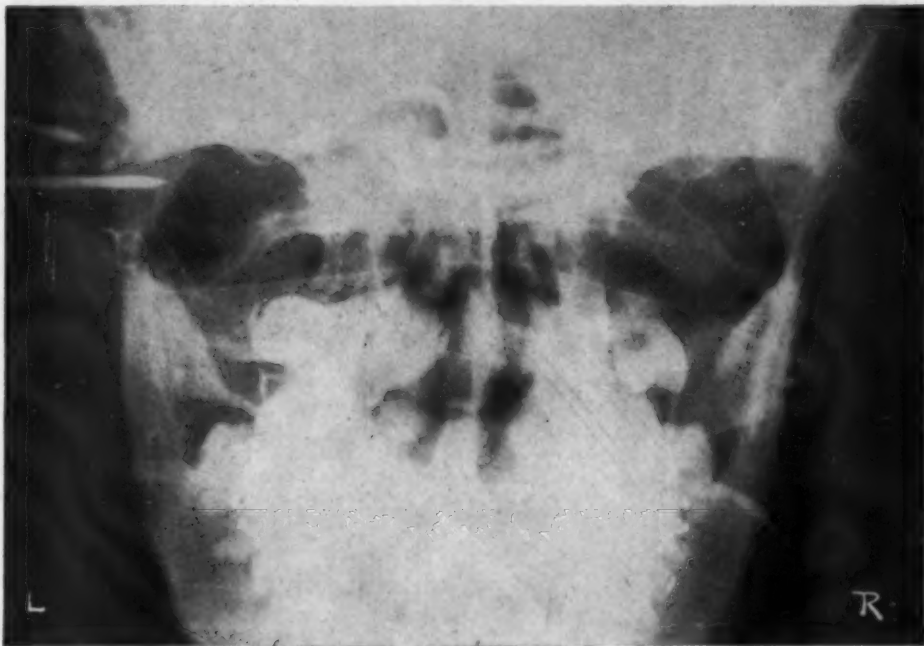


Fig. 456.—Postoperative x-ray showing skeletal and internal wiring fixation immediately after the operation.

On March 15, 1945, under pentothal intravenous anesthesia, an open reduction was performed. After the usual preparation of the temporal region, the ear, and preauricular area, an angulated vertical incision was made in front of the ear extending as far as the attachment of the lobe. The subcutaneous tissues were divided, several vessels tied, and the zygomatic process exposed. The zygomatic branch of the facial nerve was demonstrated and preserved. The joint capsule was opened and the meniscus was found in the glenoid fossa in its normal place. The condyle was located and elevated by means of two sharp hooks. Two condylar retractors were inserted to hold it in position while a hole was drilled through the neck of the condyle. The ramus was pushed into the wound by elevating the jaw, and another hole was drilled in the fractured end. A 25 gauge stainless steel wire was inserted, the fracture was properly impacted, and the ends of the wire wound tight and cut short. The head of the condyle showed a tendency to dislocate medially, and it was necessary to hold it in place by means of skeletal fixation. A half pin was inserted into the condyle just below the capsule, and another through the skin flap into the zygomatic process of the temporal bone. The two pins were fitted with links to a crossbar and this held the condyle in position. The subcutaneous tissues were closed by interrupted sutures, and the skin by a subcuticular suture. The mandible was immobilized with Jelenko splints and intermaxillary elastics, and a dressing and pressure bandage were applied.

Infection developed around one of the pins and penicillin treatment was instituted; 10,000 units were given intramuscularly every three hours. A solution of 250 units per cubic centimeter was injected into the wound after the skeletal fixation was removed on the tenth postoperative day. The infection cleared up in three days with this treatment, and the patient was discharged on April 4.

The intermaxillary fixation was removed on April 18; the patient could open her jaw moderately well at this time. She was instructed to exercise the jaw to increase the mobility of the joint. On May 31 she stated that she could open her jaw well and also extend it about $\frac{1}{4}$ inch beyond the upper teeth. A moderate Bell's sign which had developed the day after the operation—reflexes having been normal the night before—also had disappeared at this time.

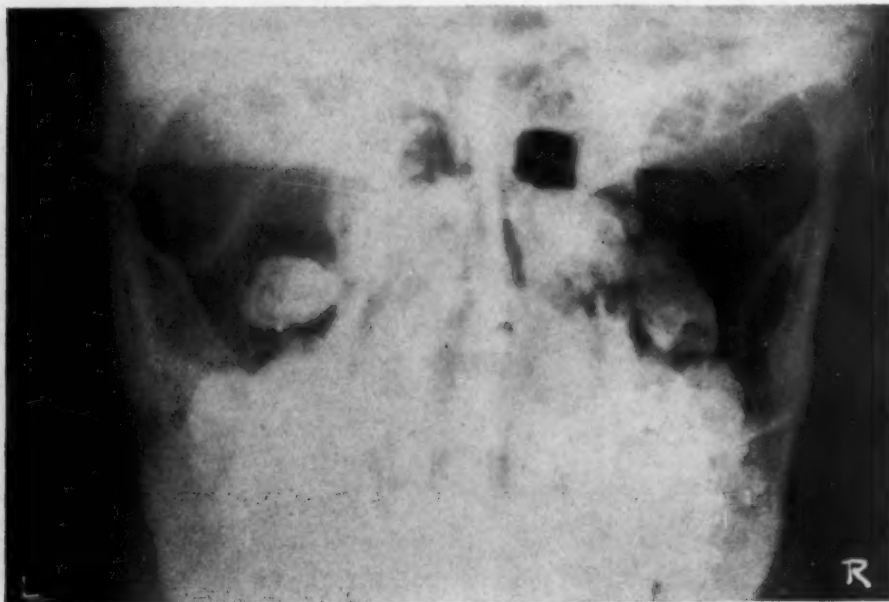


Fig. 457.—Anteroposterior view taken after removal of the skeletal fixation. The condyle is now held by interosseous wire.



Fig. 458.—Lateral view taken after removal of the skeletal fixation showing internal wiring fixation.

X-ray examination made immediately after the operation showed the reduction with wiring fixation and the pins holding the condyle in the fossa (Fig. 456). A second examination made after removal of the pins showed the position of the condyle maintained both in the anteroposterior and lateral views (Figs. 457 and 458).

Case 99

**Fracture Dislocation of the Condyle With Medial Displacement, Treated by
Internal Fixation With a Sherman Plate**

G. B. (485616), a 40-year-old man, was referred to the Dental Clinic on April 9, 1945, because of a fracture of the right jaw. This fracture had occurred three weeks previously when the patient lost his balance and fell, striking his chin on a chair.

Examination showed little evidence of the fracture. The patient could open his mouth and eat without pain, although there was crepitus on the right side in front of the tragus of the ear.

X-ray examination showed a fracture at the neck of the right condyle with dislocation to the median side (Fig. 459).



Fig. 459.—X-ray showing fracture at the neck of the right condyle with dislocation to the medial side.



Fig. 460.—Postoperative x-ray showing the fragment in good position and held by a Sherman plate.

The patient was admitted to the House on April 16, and on the next day open reduction of the fracture dislocation was performed under intratracheal gas, oxygen, and ether anesthesia. The right temporal and preauricular regions were prepared in the usual manner. An angulated vertical incision was made in front of the ear extending as far as the attachment of the lobe. The subcutaneous tissues were divided. The superficial orbital artery and vein were cut and tied, and the zygomatic process dissected down upon and demonstrated at about 2 cm. in the anteroposterior direction. The area of the temporomandibular joint was located and the capsule of the joint incised vertically. The meniscus was visible in the glenoid cavity, but the condyle was not found. After a considerable amount of exploration, it was finally located and found to be firmly attached in an inverted position to the posteromedial surface of the ascending ramus. The condyle was detached by means of a periosteotome, and by means of hooks inserted on each side it was carefully elevated into the glenoid fossa. The ramus was then located by exerting pressure in an upward direction at the angle of the jaw, and by means of retraction of the skin. About 1 cm. of the ramus could be made accessible in the wound. While the ramus was exposed, the zygomatic branch of the facial nerve was demonstrated and carefully retracted. By means of a dental drill a very small hole was drilled into the outer surface, and a Sherman plate attached to it by means of a screw. The condyle was then carefully positioned in the glenoid fossa, and its neck elevated, and the fracture reduced by the use of a bone file. While the condyle was held with a pair of subcondylar retractors, two holes were drilled into the external surface to insert the remaining screws and fix the fracture by means of the Sherman plate. After this fixation was complete it was evident that the condyle was still very slightly displaced in a medial direction, but it was impossible to make any correction since it would have been necessary to remove the plate to do it. Sulfanilamide powder was inserted, and the capsule was sutured over the head of the condyle by means of No. 00 catgut. The subcutaneous tissue was closed with catgut and the skin with a subcuticular suture. After the dressing had been applied to the wound the eye reflex was tested and found to be normal. The patient's upper denture was inserted, and the jaws were immobilized by applying nasal wires through the piriform apertures and fastening them to a bar wired to the lower teeth.

The patient received an intravenous infusion of 500 c.c. of 5 per cent dextrose in water on the day of the operation, and 1,000 c.c. the next day. He was also given 32,000 units of penicillin on the day of operation, and 96,000 each day for the next seven days.

The postoperative x-ray showed the fragment held in good position by screws and the metal bar (Fig. 460).

On April 18 the drain was removed and the wound appeared satisfactory. The dressing was changed; there was some oozing of blood from the left nostril. The eye reflex, which had been normal the night after the operation, showed slight paralysis. The patient was discharged on April 25 to be followed in the Outpatient Department.

The patient's progress was satisfactory; the wires were removed on May 24. Two weeks later he had good motion of the jaw in all directions and was instructed to exercise the jaw to open the bite a little wider in the incisor region. The eye sign had almost disappeared. The patient was discharged to return in one month for a check-up examination.

SUMMARY

The causes, diagnosis, treatment, and prevention of complications following the treatment of condylar fractures have been described. Five cases presenting major functional disturbances are reported.

The use of various positions in x-ray examination has been recommended in order to obtain a clear picture of the condition present.

The prevention of complications includes exact reduction by the open method and internal wiring, plating, or skeletal fixation. In fractures with displacement, interosseous wiring gives excellent results; in fracture dislocation skeletal fixation of the condyle to the temporal bone is recommended in addition to the wiring. The two methods may be replaced by the use of the Sherman plate. Two cases of fracture dislocation illustrating the two methods are presented.

HYPEROSTOSIS OF THE MANDIBULAR CONDYLE

WITH REPORT OF TWO CASES

KURT H. THOMA, D.M.D.

OVERGROWTH of the mandibular condyle is a very rare disease. A few cases have been reported under the names of osteoma, osteochondroma, and hypertrophy of the condyle. Gruca and Meisels (1926)* reviewed the literature, collecting fourteen cases and adding three of their own; Ivy (1927)† has reported three cases. The oldest case on record is that reported by Eckert (1899)‡; it has been mentioned in Scudder's *Tumors of the Jaws*,§ and other publications. In all these cases a slowly developing deformity of the face was the main complaint; secondary disturbances were malocclusion, and in some cases slight interference with motion of the jaw.

The etiology is not clear. In most cases no definite cause has been discovered, but the suggestion has been made that middle ear infection may stimulate an inflammatory hyperplasia and overgrowth of the epiphysis. One of Ivy's cases presented a cystic overgrowth, but this patient had no visible deformity.

Some time ago, I (1936)|| reported the case of a 10-year-old boy with generalized osteomyelitis in which the ramus of the mandible was involved through hematogenous channels. The effect of the inflammatory process on the enamel organ of the second molar caused the formation of a cyst which pushed the third molar into the coronoid process of the mandible. The cyst and the two teeth were removed in 1935 with some difficulty, because of moderate ankylosis of the jaw. The healing was uneventful. The x-ray taken at that time showed a slightly enlarged condyle; it contained osteolytic areas which were interpreted as osteomyelitis (Fig. 461). Because of a number of serious episodes involving various bones (tibia, fibula, humerus, and ulna), his parents did not consent to an operation to relieve the ankylosis. The patient was seen again in December, 1944, because of a pulp infection in a molar tooth. He was unable to open his mouth far enough for direct examination. The x-ray showed extensive caries destroying practically the entire crown of the tooth. The joint on the side where the infection and cyst had occurred was x-rayed at this time, and a greatly enlarged, irregularly deformed condyle was found, which caused the ankylosis (Fig. 462). This case is an illustration of an inflammatory enlargement of the condyle. It caused no overgrowth of the affected mandible; contrarily, the involved side was underdeveloped, as it generally is in patients whose jaws have been immobilized because of ankylosis. This phenomenon has been discussed by me previously (1938).¶

*Gruca, A., and Meisels, F.: Asymmetry of the Mandible From Unilateral Hypertrophy, *Ann. Surg.* 83: 755, 1926.

†Ivy, R. H.: Benign Bony Enlargement of the Condylod Process of the Mandible, *Ann. Surg.* 85: 27, 1927.

‡Eckert, B.: Osteoma des Unterkiefers, *Beiträge z. klin. Chir.* 23: 676, 1899.

§Scudder, C. L.: *Tumors of the Jaws*, Philadelphia, 1915, W. B. Saunders Co.

¶Thoma, K. H.: A Case of Generalized Osteomyelitis With Interesting Jaw Involvement Leading to Formation of Parodontal Cyst, *INT. J. ORTHODONTIA* 22: 516, 1936.

||Thoma, K. H.: Principal Factors Controlling Development of Mandible and Maxilla, *AM. J. ORTHODONTICS AND ORAL SURG.* 24: 171, 1938.

The two cases to be reported in this paper, however, are of a different nature. There were no etiological factors disclosed by studying the histories of the patients. The patient's face in both cases was markedly asymmetrical. This asymmetry consisted, first, of an elongation of the ramus which lowered the angle of the jaw and pushed the chin to the other side (Figs. 463 and 472),

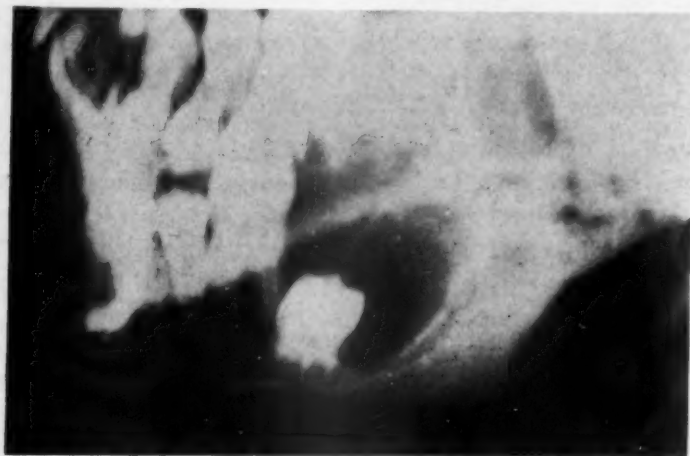


Fig. 461.—X-ray of the jaw of a patient with generalized osteomyelitis showing enlargement of the condyle.

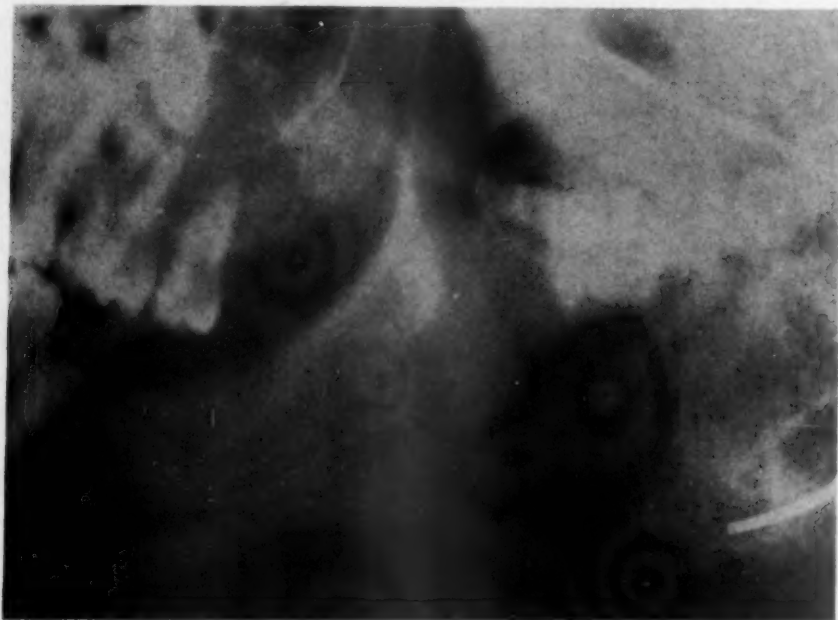


Fig. 462.—X-ray of the jaw of the same patient nine years later showing ankylosis of the condyle.

and second, of a secondary deformity of the temporal region, zygoma, and zygomatic arch. In one case there was considerable interference with the mobility of the mandible; in the other case the mobility was not greatly interfered with. In the former, the teeth, such as they were, occluded; in the latter, the last molars had elongated and were in contact when the mouth was closed, but the first molar and premolar areas were affected by marked open-bite. Apparently the lengthening of the ramus produced a compensatory enlargement of the tuberosity (Fig. 475).

Case 100

Hyperostosis of the Mandibular Condyle

H. L. (479040), a 37-year-old man, was admitted to the hospital on Feb. 7, 1945, with a tumor of the jaw in the area of the left condyle.

The patient had had the tumor since he was a child, but did not know whether or not it was present at birth. It had increased in size slightly in recent years, and had caused some pain. The pain first occurred about twelve years before and was associated with discharge of pus from the auditory canal. The latter had always been somewhat occluded, and the patient was deaf in the left ear. Since then there had been several recurrences of pain with discharge from the ear and inflammation and swelling both in front of the ear and on the mastoid



Fig. 463.—Hyperostosis of the mandibular condyle causing swelling of the side of the face and deviation of the chin.



Fig. 464.—Photograph showing limit of mandibular motion and malocclusion produced by the deformity.

process. The patient said these attacks were usually precipitated by a cold or "getting too hot," and the pain had been severe enough to keep him awake at night.

Twelve years ago at the time of the first attack of pain, a local doctor attempted to remove some of the bone through the auditory canal under local anesthesia, but was forced to stop because of pain. There was some symptomatic relief, however, and no further treatment had been attempted. Recently, the patient noticed increasing difficulty in opening the mouth more than one inch. His chin was drawn to one side. There had also been a dull pain in the jaw during the past year which was different from the usual acute attacks of pain. The past history was otherwise negative except for removal of a bladder stone about three years before.

Examination showed a large swelling on the left side of the face, distorting it and pushing the chin to one side (Fig. 463). A large, very hard, nonmovable mass, approximately 6 by 6 cm., was present in front of the ear. The teeth did not occlude well, and the left mandible was lower than the right. Hearing in the left ear was poor. The external canal was moderately compressed by an extrinsic mass pushing on it. In the lower jaw on the left, the second incisor, canine, and first premolar were the only teeth present; there were six teeth on the right; in the upper jaw all the teeth were present (Fig. 464).

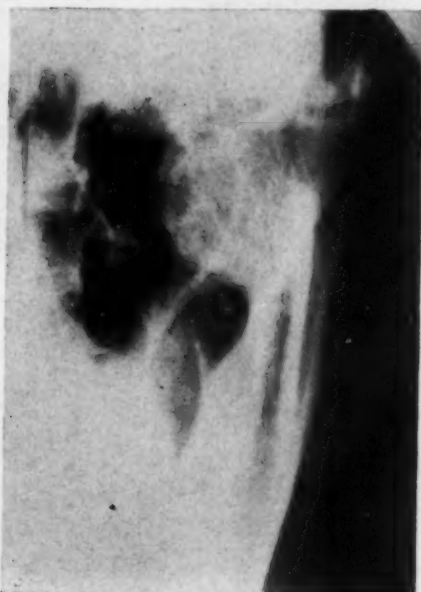


Fig. 465.



Fig. 466.

Fig. 465.—Anteroposterior view of the left temporomandibular region showing marked hyperostosis of the condyle and calcification of the sphenomandibular ligament.

Fig. 466.—Lateral view showing distorted condyle and unerupted third molar, over which there is a bony spur.

X-ray examination showed considerable deformity of the left jaw due to marked thickening of the bone in the base of the condyloid process of the mandible. There was a fusiform thickening of this area of the jaw the width of about 3 cm. It showed fairly normal bone structure with a few cystlike areas of bone rarefaction (Fig. 465). The left side of the mandible seemed elongated and somewhat pushed outward. There was a spurlike pointed projection of bone at the inner side of the mandible in the third molar area; this also consisted of normal bone structure. The left lower third molar was impacted and carious and there was an area of bone rarefaction extending from the carious crown and the alveolar margin of the bone which had the appearance of infected bone. (Fig. 466). There were also abscesses surrounding the left canine and the first premolar. The right side of the mandible appeared fairly normal. The findings were those of a deformity of the left jaw either due to developmental changes or an osteoma involving the basal portion of the condyloid process.

In consultation with the Ear Clinic, it was found that the patient had conduction deafness; the canal was completely occluded. X-rays of the mastoids showed a normal right mastoid, and a left with increased density and marked sclerosis.

On February 13, under endotracheal gas, oxygen, and ether anesthesia, a partial excision of the osteoma was performed. The skin of the temporal region, the cheek, and the ear,

was prepared in the usual manner. Two cubic centimeters of monocaine-epinephrine were injected at the site of the incision in order to produce hemostasis of the subcutaneous tissues. An angulated vertical incision was then made in front of the ear and extended as far down as the lobe. The subcutaneous tissue was divided and found to be at least twice as thick as normal. The zygomatic arch was then demonstrated and the periosteum incised until the area of the temporomandibular joint could be discovered. The posterior attachment of the masseter muscle was detached from the zygomatic arch. The capsule was incised and the condyle laid bare (Fig. 467). The anesthetist was asked to move the patient's jaw, and it was noticed that there was practically no motion in the joint itself. There seemed to be no bony tumor



Fig. 467.—Vertical incision in the preauricular area showing the lateral aspect of the hyperostosed condyle.

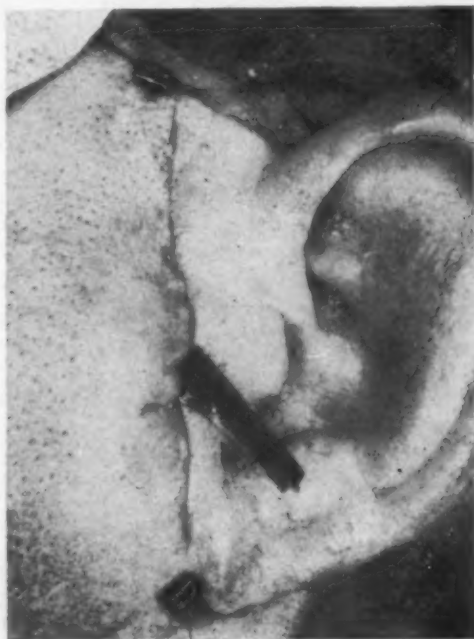


Fig. 468.—Photograph showing the incision closed with subcuticular sutures and a drain inserted to prevent a hematoma.

visible, but deformity of the entire area was noticed. The ramus was dissected down upon as far as the facial nerve would allow, and after it was exposed an osteotomy was performed in this region. First, holes were drilled by means of dental burrs, and it was noticed that the latter were not long enough to penetrate the thickness completely. Osteotomes were then used to complete the osteotomy, and the bone was found to be extremely hard and difficult to cut. Finally the osteotomy was completed so that the ramus could be moved by moving the mandible. An osteotome was then inserted into the joint space and driven in about 1 cm. by means of a mallet. With leverage action the bone was separated from the glenoid fossa, and a large bony mass removed after carefully detaching it from its surrounding tissue. When examined it was found to have a smooth, hard surface which was slightly lobulated (Fig. 469). A large space resulted where the bone had been removed, and careful investigation after the bleeding had been stopped with adrenalin packs disclosed that there was still a large part of the tumor left attached to the inner surface of the mandible. This, however,

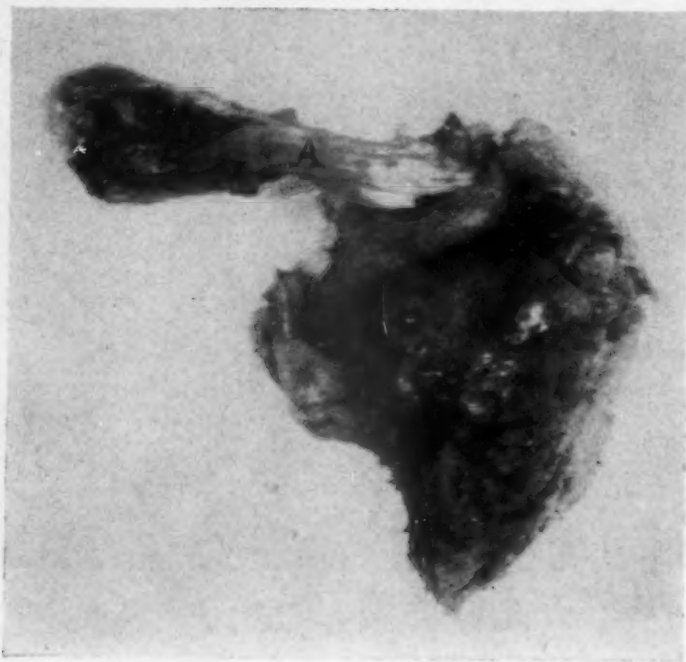


Fig. 469.—Excised part of the hyperostosed condyle with the external pterygoid muscle, A, attached.



Fig. 470.—Postoperative anteroposterior x-ray after excision of the part of the condyle extending into the glenoid fossa. The osteotomy line is indicated by dots.

could not be reached very well, since it was impossible to dissect further down on the ramus. As the patient had very good motion and would probably get relief of his symptoms, including pressure on the external auditory meatus, it was felt that nothing more should be done at this time until the situation could be further appraised by x-ray examination. Fibrin foam and thrombin were placed on the bleeding bony surface where the osteotomy had been performed, and were also used to fill in some of the space created by removal of the bony mass. The subcutaneous tissues were closed with catgut sutures. A rubber-dam drain was inserted to prevent hematoma from forming. The skin was closed with a subcuticular suture (Fig. 468). One tooth was extracted on the left side of the mandible because of apical infection.

The patient received an intravenous infusion of 1,500 c.c. dextrose in water on the day of operation, and 1,500 c.c. dextrose in saline on the first postoperative day. He received 2.5 Gm. sulfadiazine on the day of operation and 4 Gm. each day for the next seven days. On February 8 the white cell count was 7,700 and the hemoglobin 17 Gm.; on February 17 the white cell count was 6,500 and the hemoglobin 16.5 Gm.

Postoperative roentgen examination showed the removal of the upper part of the tumor; a large lobe was still found extending medially from the ramus (Fig. 470).

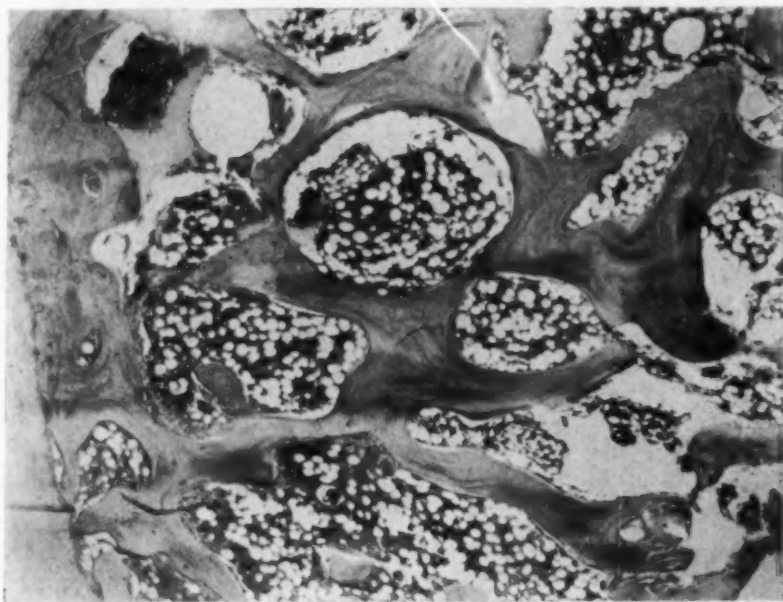


Fig. 471.—Photomicrograph of excised condyle shows normal bone; no specific disease process could be found.

It was noted that the masseter and internal pterygoid muscles elevated the jaw, closing the space between the base of the skull and the cut-off tumor mass. This caused the upper molars to traumatize the mandibular ridge, and the gingiva became irritated and painful. This condition was corrected on February 19 when the upper left second and third molars were extracted under novocain infiltration anesthesia. This removed the pressure on the lower gingival tissue and improved the occlusion.

On February 21 the wound was healed and the subcutaneous suture was removed. Movement of the jaw was much improved, and there was some relief of the pain, but the facial disfigurement was only slightly corrected. The consensus of opinion was that complete removal of the osteoma might result in more severe interference of the occlusal relationship of the jaws, and was therefore abandoned. The patient was discharged on February 21 to his local doctor.

Pathologic examination: A piece of angulated bone was received measuring approximately 4 by 15 cm., with some soft tissue attached, probably part of the external pterygoid muscle (Fig. 469). Grossly the bone was normal. No diagnostic abnormality was recognized. The microscopic findings revealed no disease; a part of the section is shown in Fig. 471.

On March 26 the patient's doctor reported that the patient did not have any great improvement in the external deformity, but that he could open the mouth wide and chew without difficulty. For the past few days he had been relieved of the pain, which was most important. The patient stated that there was little improvement in hearing in the left ear, but that the operation had been beneficial in that it relieved the pain and enabled him to chew normally.

Case 101

Hyperostosis of the Mandibular Condyle

J. C. consulted me on June 5, 1945, on recommendation of his physician, regarding a deformity of the jaws. His dentist furnished models of his jaws and x-rays of the teeth which showed an abnormal relationship of the jaws with open-bite on the left side of the face.

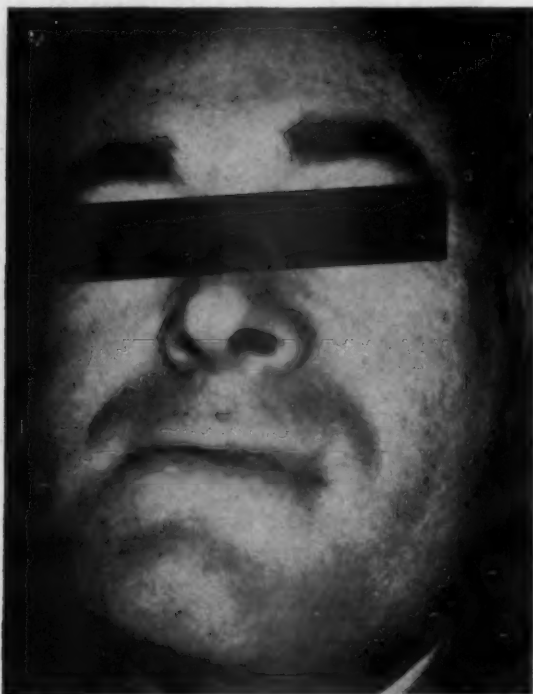


Fig. 472.—Patient with hyperostosis of the mandibular condyle presenting facial asymmetry.

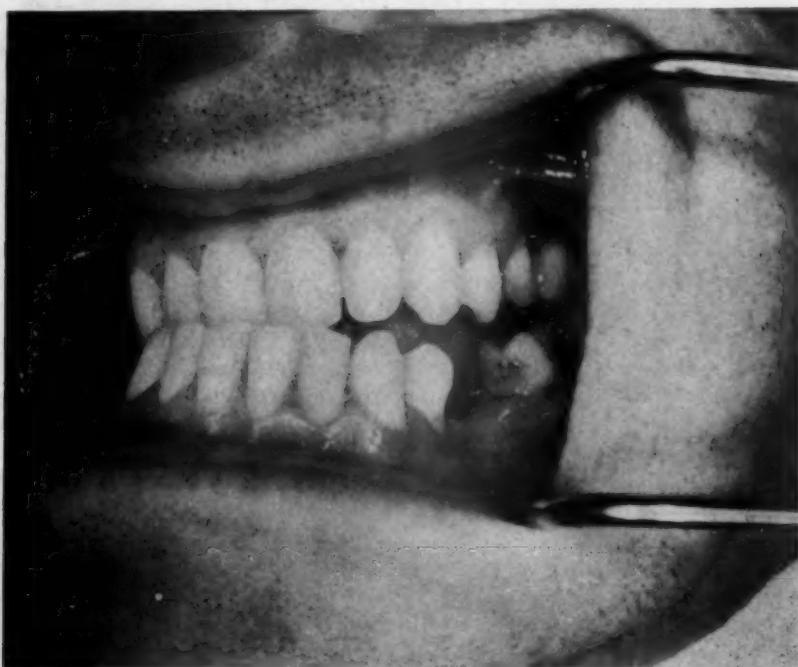


Fig. 473.—Malocclusion caused by hyperostosis of the mandibular condyle.

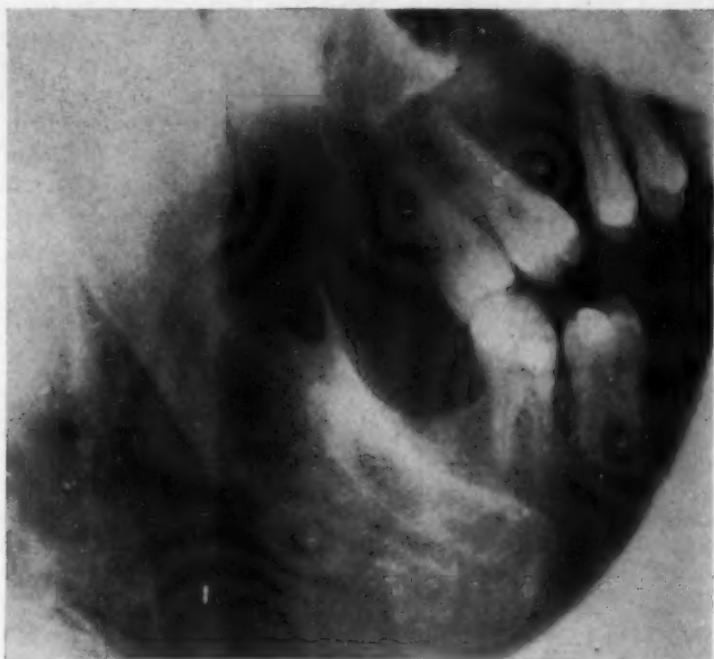


Fig. 474.—Lateral x-ray view showing elongation of the ramus with an osteolytic defect in the anterior border.



Fig. 475.—X-ray showing asymmetry of the skull caused by hyperostosis of the left mandibular condyle.

When the patient was examined, he stated that he had had a facial deformity since he could remember, and demonstrated this by old photographs. A marked swelling of the preauricular area was noted on the left, the left angle of the jaw was found to be much lower than the right, the chin was turned down and to the right, and the corner of the lip on the left was lower than that on the right (Fig. 472). Examination of the occlusion of the teeth showed contact of the second molars on the left, but the remaining teeth on that side failed to occlude. He also had a reversed overbite (Fig. 473). The mobility of the mandible was not affected; he had good hinge as well as lateral or sliding motion, and could open his jaws almost the normal extent.

The roentgen examination in lateral view showed the open-bite and an elongated, narrow vertical ramus with normal development of the coronoid process, but an osteolytic defect in the anterior border (Fig. 474). The anteroposterior view disclosed a marked overgrowth of the left condyle (Fig. 475).

Since the condylar deformity did not produce any functional disturbances, the patient was advised that the malocclusion could be corrected by a bilateral mandibular osteotomy. He was not interested in an improvement of the relationship of the jaws, however, but desired an operation to correct the swelling on the side of the face. The only operation that could be performed was to remove the enlarged condyle, but this would not alter the facial asymmetry which was due to secondary deformity of the temporal and zygomatic bones. Therefore, an excision of the condyle would not insure a good cosmetic result. On the other hand, the function of the jaw after operation might be adversely affected. The patient was advised against excision of the tumor, and to have the occlusion improved by prosthesis.

DISCUSSION

The first patient presented a debilitating deformity and symptoms which required an operation. Partial excision appeared to be justified since it mobilized the jaw without producing too much occlusal difficulty. It was necessary to extract the upper molars in order to allow the shortened ramus to be pulled up by the elevator muscles. It should be pointed out that in this case the muscles were under tension, which is not the case in an ordinary ankylosis where the ramus is of normal size and condylectomy causes no occlusal alterations. The operation did not improve the facial asymmetry to any great extent since the deformity involved the entire half of the facial skeleton, which changed to compensate for the ever-growing condyle.

The second patient was advised that the facial asymmetry could not be improved by operation, as it was of a similar nature to that in the first case. Since there was no difficulty in articulation, a bilateral osteotomy was suggested to correct the interrelationship of the jaws. This was refused by the patient as he was interested only in an operation in the condylar region to correct the temporomandibular asymmetry. Since excision of the enlarged condyle would not guarantee improvement in appearance, and functional improvement was not necessary, I discouraged excision.

These two cases have one interesting factor in common. In both patients, the elongation of the jaw was due partly to the enlargement of the condyle, and partly to the unusual growth of the ramus itself. This indicates clearly that the growth was governed by the condylar epiphysis. This is also borne out by the fact that the coronoid process, especially in the second case, has not participated in this growth. If the development of the ramus had been of the interstitial type, both processes would probably have participated and would be found at a level comparative to that of the normal jaw. A similar condition was also observed by Ivy in one of his cases.

No cause could be discovered in either of these cases. The suggestion that a middle-ear infection may stimulate overgrowth of the epiphysis is of interest, because the first case presented such a condition. I feel that in my case the

aural symptoms were decidedly secondary in nature due to pressure exerted by the tumor and occlusion of the auditory canal which occurred long after the onset of the disease.

The enlargement of the condyle in both cases was in medial and lateral directions; the anteroposterior dimension was not altered to any great extent. The specimen had the appearance of a thick watch rather than that of a globe. Histologic examination of the excised part showed no evidence of inflammation or of tumor formation, although it might be classified as an osteoma because of the lobulated surface.

FURTHER USES FOR THE PERIPHERAL BONE CLAMP

KURT H. THOMA, D.M.D., MARTIN WENIG, D.D.S., AND
SAMUEL I. KAPLAN, D.D.S.

THE use of a new appliance, the peripheral bone clamp, has been described in Volume IV of the Clinic of the Massachusetts General Hospital. This appliance was developed by us to be used at the angle of the jaw as a substitute for the half pins which have been found unsatisfactory in many cases in the thin bone of the mandibular ramus. Since then, clamps have been used to advantage in a number of fractures at the junction of the vertical and horizontal rami, in combination with two half pins in the anterior fragment, and a Frac-Sure connecting bar.

New applications for this appliance have been discovered, and will be presented in three case reports illustrating their use, as follows:

1. For skeletal fixation of a fracture which was thought to require more time to heal than a simple fracture, because it extended through a mandibular cyst. It healed uneventfully in about six weeks.

2. For fixation of the two fragments of an old ununited fracture treated by a bone graft, requiring immobilization for eight weeks or longer. In this case two clamps were used, one at the angle of the vertical ramus, the other at the anterior part of the horizontal ramus. It was believed that the clamps would be tolerated longer than pins around which resorptive changes are frequently seen, making their removal advisable after five weeks of immobilization. It was thought that the clamps would hold the fracture fixed as long as necessary, and would be effective even if the mandibular immobilization was terminated before that time.

3. For direct fixation of an oblique fracture through an edentulous section of the mandible. It was reduced by an open operation and immobilized by placing a clamp directly over the fracture site. Intraosseous wiring is not satisfactory in oblique fractures; it tends to cause overriding. When one clamp is placed over the fracture site, the fracture can be perfectly impacted and completely immobilized. The pin which fastens the clamp, not being needed for the attachment of other parts of the Frac-Sure appliance, can be cut short and covered with a dressing wound around and over it and held by adhesive tape.

Case 102

Fracture of Mandible and Cyst Immobilized by Clamp and Pin Fixation

G. L. (33604), a 42-year-old man, came to the Emergency Ward on Jan. 27, 1945, with a painful and swollen jaw. He had received a blow four days before.

Examination showed mild tenderness along the lower margin of the left mandible near the angle. The teeth, most of which were very loose, did not meet in normal occlusion. There were pain and a grating noise when he closed the mouth with force. There was a past history of duodenal ulcer and chronic alcoholism.

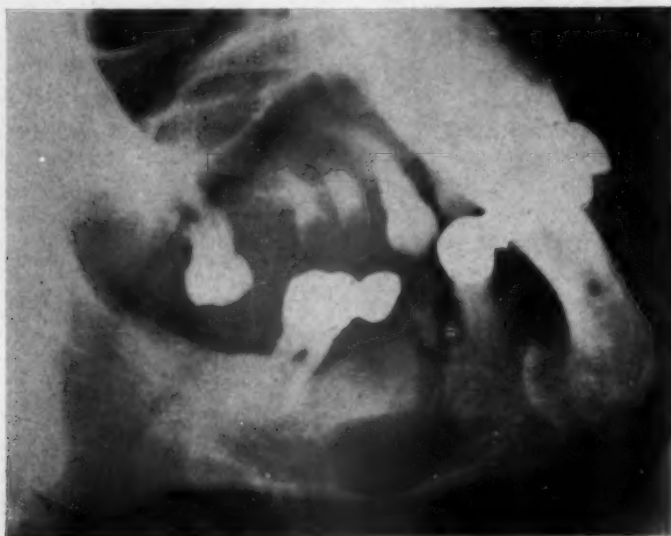


Fig. 476.—Lateral view of jaw showing fracture of the mandible through a radicular cyst.

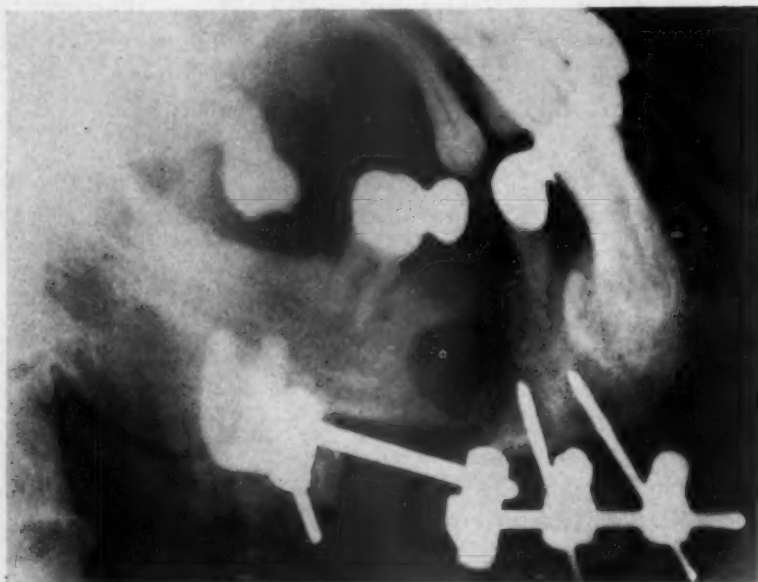


Fig. 477.—Postoperative x-ray showing immobilization with a peripheral bone clamp and pin fixation.

The x-ray examination revealed a fracture of the horizontal ramus of the left mandible. The fracture line passed through the socket of an infected second premolar and a radicular cyst. There appeared to be several infected roots (Fig. 476).

Diagnosis: Fracture of the left mandible complicated by a cyst. Skeletal fixation was advised since the teeth were not suitable for intermaxillary fixation.

On January 30 excision of the cyst and reduction of the fracture were performed under endotracheal gas, oxygen, and ether anesthesia. The skin was prepared in the usual manner

and the oral cavity with zephiran. An incision was made on the outer surface of the alveolar process exposing the bone. A broken-down root of a premolar tooth was removed; its socket was found to communicate with the cyst. A window was cut in the outer surface of the mandible, and this cyst sac detached by means of a periosteal elevator. It was removed in one piece, and the mandibular artery and nerve could be seen lying in the floor of the cavity. Sulfanilamide powder was dusted into the cavity, and fibrin foam and thrombin were inserted. The mucosa was replaced and the incision closed with sutures.

With a new sterile setup the operation was continued. An incision was made at the angle of the jaw about 1 cm. below the inferior border. The subcutaneous tissue and platysma were divided, and the angle of the jaw was exposed. Here the masseter muscle on the outside and internal pterygoid muscle on the inside were detached for a short distance in order to attach the peripheral clamp. This was fitted in place, and, after a stab incision had been made in the skin, the pin was inserted and tightened. This gave firm attachment to the posterior fragment. Two half pins were inserted into the anterior fragment through the stab incision after holes had been made with a drill. The remainder of the Frac-Sure appliance was then attached, the cross links and a connecting bar to fix the fracture after it had been properly reduced. The incision was closed and a Barton bandage applied for temporary immobilization of the jaw.

The patient received sulfadiazine, 2 Gm. to start and 1 Gm. every four hours, for five days postoperatively. On January 31 the white cell count was 12,000 and the hemoglobin 73 per cent. Recovery was uneventful. The sutures were removed and the patient discharged on Feb. 5, 1945, to be followed in the Outpatient Department.

Pathologic report showed a soft, red-gray tissue, a cyst membrane with chronic inflammation and fibrosis.

Roentgen examination showed a satisfactory postoperative result. In an attempt to impact the fracture, it was slightly telescoped (Fig. 477). It was felt that this would not cause any debility since the patient's teeth were all loose and should be removed.

On March 14 the fracture was tested. There was slight motion although it seemed to be getting firm. Healing of course was complicated by the cyst. On April 2 the fracture seemed firm, and it was decided to remove the appliance. Three days later, under novocain anesthesia and after the usual preparation of the skin, the pins were removed. Following this an incision was made over the clamp at the site of the scar of the former operation. The clamp was removed and the subcutaneous tissues closed with catgut and the skin with a subcutaneous suture. One week later the wound was healing well and the suture was removed. The patient was referred to the Dental Clinic for extraction and restoration of his teeth.

Case 103

Nonunion of Fracture of the Mandible With Rib Graft and Immobilization by Skeletal Fixation With Clamp Attachments

J. J. (488672), a 43-year-old man, was referred to the Dental Clinic on May 2, 1945, with nonunion of a fracture of the mandible.

Nine months previously, the patient had been in a fight and was struck on the jaw, sustaining a fracture at the angle of the mandible on the right. The fracture was reduced first by a face mask, and when this proved unsuccessful, the jaw was immobilized by inter-maxillary wiring by his local dentist, after a third molar which complicated the fracture was removed. The fracture, however, did not unite and there was no evidence of callus formation in the x-ray picture. The jaw was very uncomfortable and the patient could not chew food; he had lived on a liquid diet since the accident.

X-ray examination showed a gap in the bone of the horizontal ramus of the right lower jaw, somewhat irregular in outline, about 1 cm. in size. The apposition of the bone was satisfactory, but the adjoining surfaces of the fragments appeared to be smooth (Figs. 478 and 479).

Jelenko splints were applied to the teeth, and the patient was admitted to the House on May 16 for a rib graft to the ununited fracture. The right mandibular area was shaved and scrubbed, and covered with a sterile dressing the day of admission and again the night before the operation. The donor site, the anterior chest area, was also shaved and scrubbed.

On May 18, under endotracheal gas, oxygen, and ether anesthesia, the skin on the right side of the face was prepared in the usual manner and an incision was made about 1 cm. below

the inferior border, extending from the region of the mental foramen beyond the angle of the jaw (Fig. 480). The skin, subcutaneous tissues, and platysma were divided. The external maxillary artery and vein were tied and cut (Fig. 481). By blunt dissection the mandible was exposed at the inferior border. The fracture site was located and a great deal of venous bleeding was encountered. After the periosteum was incised, the bone was laid bare on the internal and external surfaces about halfway up in a vertical direction (Fig. 482). Scar tissue

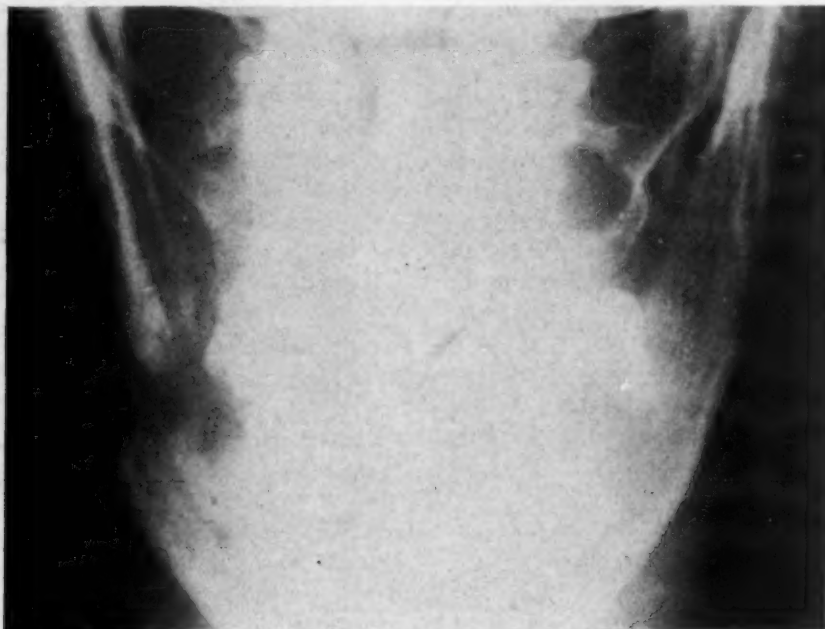


Fig. 478.—X-ray showing ununited mandibular fracture, anteroposterior view.

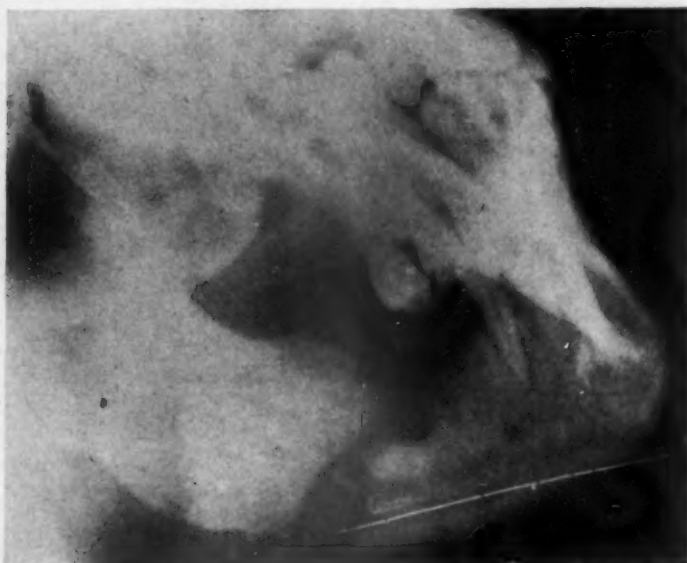


Fig. 479.—Lateral x-ray showing ununited mandibular fracture.

between the fragments was removed, and the bone was found to be eburnated on the fractured ends (Fig. 483). Since no perforation was made into the mouth, it was thought best to remove the rib at this time. The surgical team removed a piece of the sixth rib (Figs. 484 and 485) of the appropriate length and split it lengthwise. The wound over the jaw, which had been packed with a wet saline sponge, was now opened, and by means of rongeur forceps the two ends of the fragments were freshened. Two peripheral clamps were inserted, one near the mental foramen and the other posterior to the angle of the jaw. These were fastened by

means of a pin inserted through a separate stab incision in the skin. The outer surfaces of both bone fragments were decorticated over an area to correspond with the width of the rib (Fig. 486). The split rib was then inserted and fitted into the decorticated areas as accurately as possible. Holes were drilled with a dental drill through the graft and the jaw, and the rib was fastened to the jaw with 25 gauge stainless steel wires inserted in the anterior fragment horizontally (Fig. 487), and in the posterior fragment vertically (Fig. 488). Another piece of rib was then fitted between the two fragments on the lingual side of the jaw.



Fig. 480.—Incision for bone graft of the mandible.

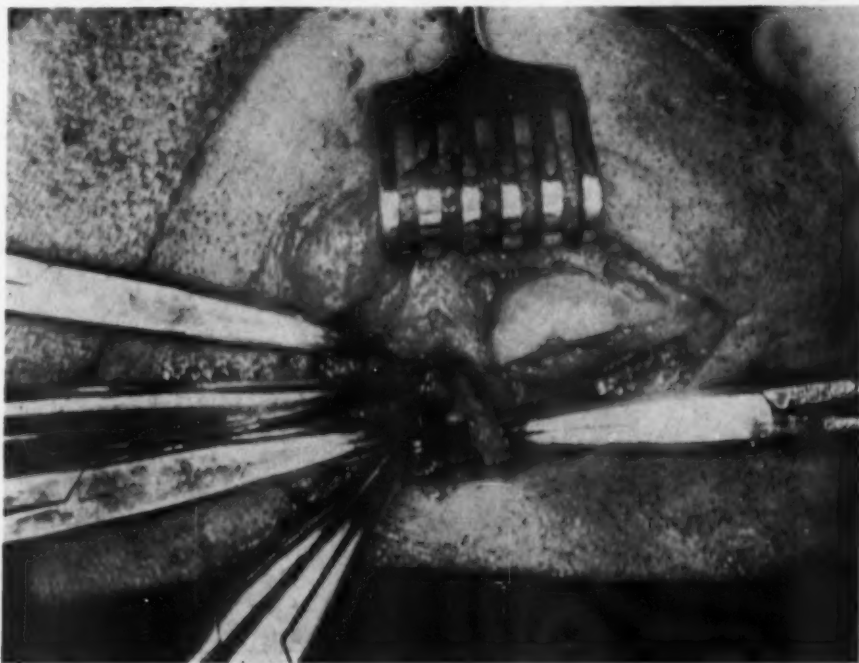


Fig. 481.—Demonstration of external maxillary artery.

The subcutaneous tissue was closed with interrupted catgut sutures and the skin with interrupted Dermalon sutures. Then the crossbar of the Frac-Sure appliance was applied to the two clamps by means of links placed on the pins; this immobilized the fracture completely. The mandible had been previously immobilized by means of wires attached to the lugs of the Jelenko splints. A bulky dressing was applied to the face and held in place with an elastic bandage.

The patient received an intravenous infusion of 1,500 c.c. of dextrose in water after the operations, and also on the next day. He was given penicillin, 72,000 units on the day of operation, and 96,000 units on each of the following ten days. On the first postoperative day there was some swelling of the face, but otherwise the patient's condition was good.

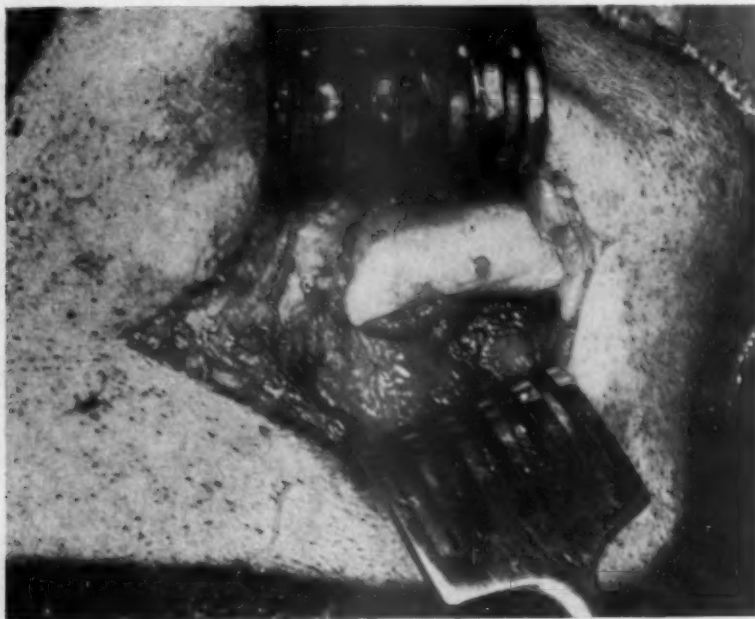


Fig. 482.—Exposure of the anterior fragment.

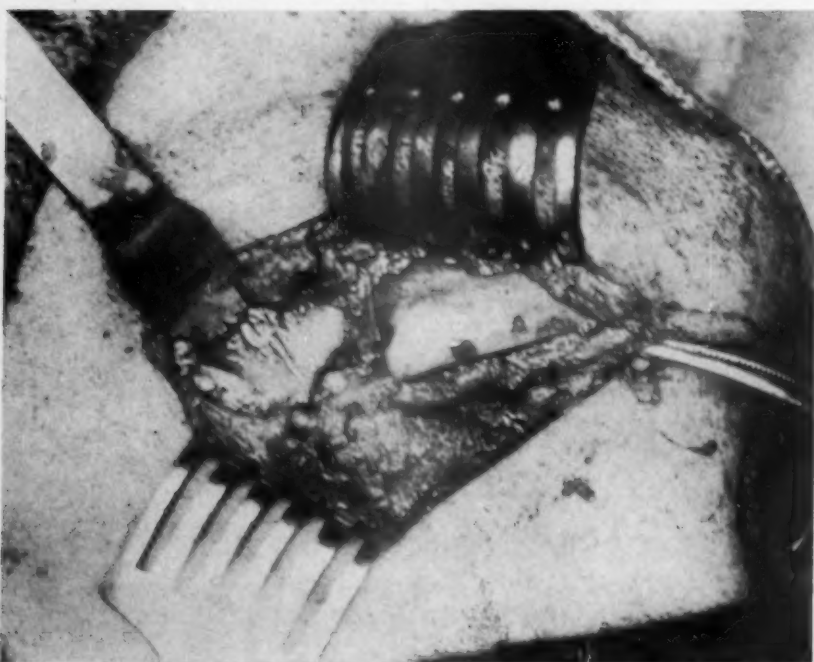


Fig. 483.—Scar tissue between the fragments has been removed.

The chest was clear and there was no emphysema noted. On May 25 the stitches were removed from the chest and from the jaw, and small dressings were applied around the pins to protect the stab incisions (Fig. 489). He received a high-calorie liquid diet with 5 c.c. of vitamin B daily.

X-rays taken postoperatively showed the bone fragments held in place by a metal splint (Fig. 490). A bone graft fixed to the adjoining portion of the right lower jaw was seen, and

held in place by metal sutures. The apposition appeared satisfactory. A throat stick fixed to a bandage was placed around the patient's head when lying down to prevent him from rolling onto the operated side and disturbing the external fixation (Fig. 491).

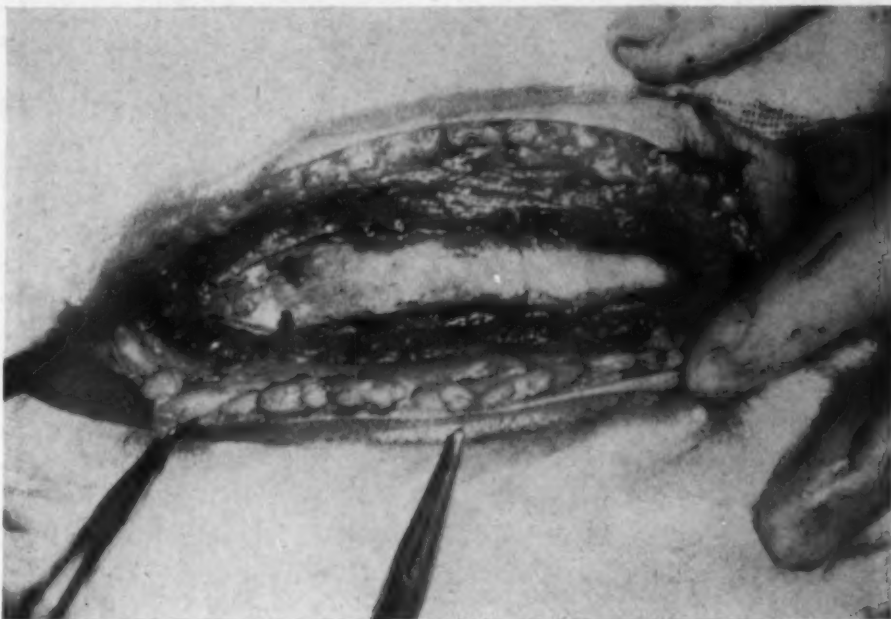


Fig. 484.—Photograph showing exposure of the rib.

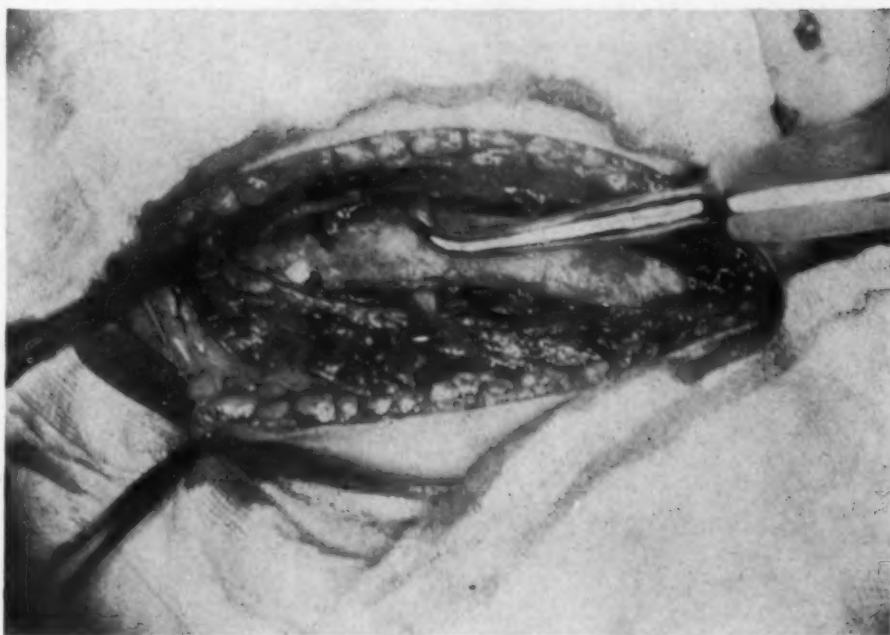


Fig. 485.—Removal of the rib to be transplanted.

The patient was discharged on the thirteenth postoperative day to be followed in the Outpatient Department. When he was seen one week later, he was doing very well. There was no seepage around the pins and no reaction from the clamps. The intermaxillary fixation was discontinued after six weeks of immobilization. At this time the patient had slight muscular trismus. The external clamp fixation seemed to hold the fracture immobilized. At the end of the eighth week the clamps were removed under pentothal intravenous anesthesia. Incisions were made over each clamp exposing it so it could be grasped with a pair of bone forceps. After the pin was removed, the clamp was disengaged on the inner surface of the jaw



Fig. 486.—Peripheral bone clamps inserted at the angle of the jaw and in the anterior region, and decortication of the fragments.

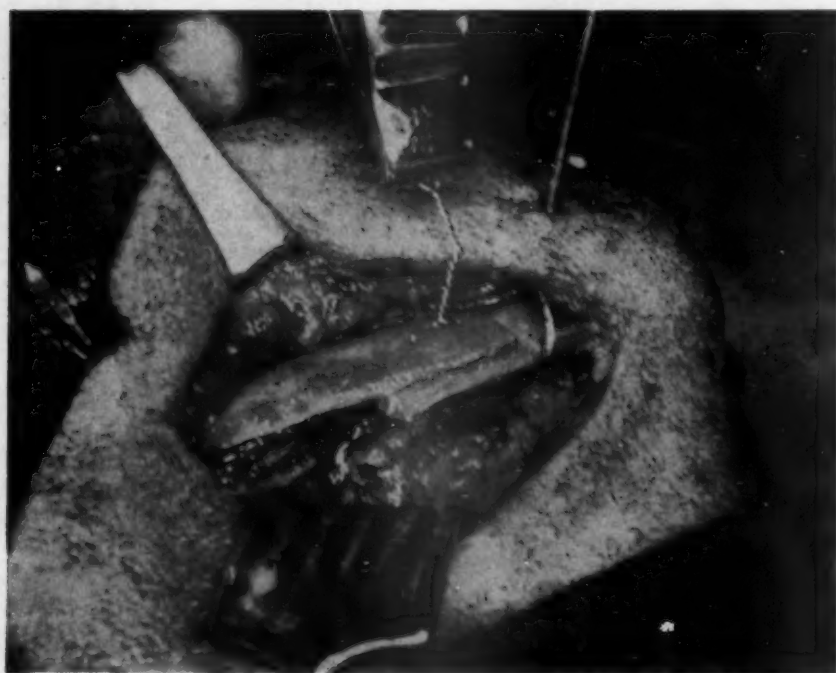


Fig. 487.—Bone graft fitted to the decorticated area and attached by means of a wire to the anterior fragment.

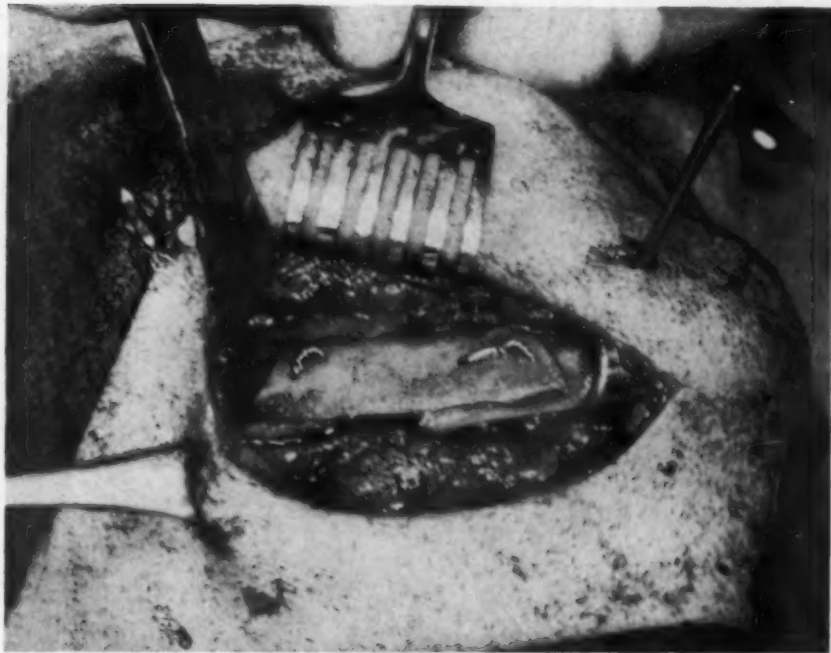


Fig. 488.—Bone graft attached to the anterior and posterior fragments by means of steel wire.

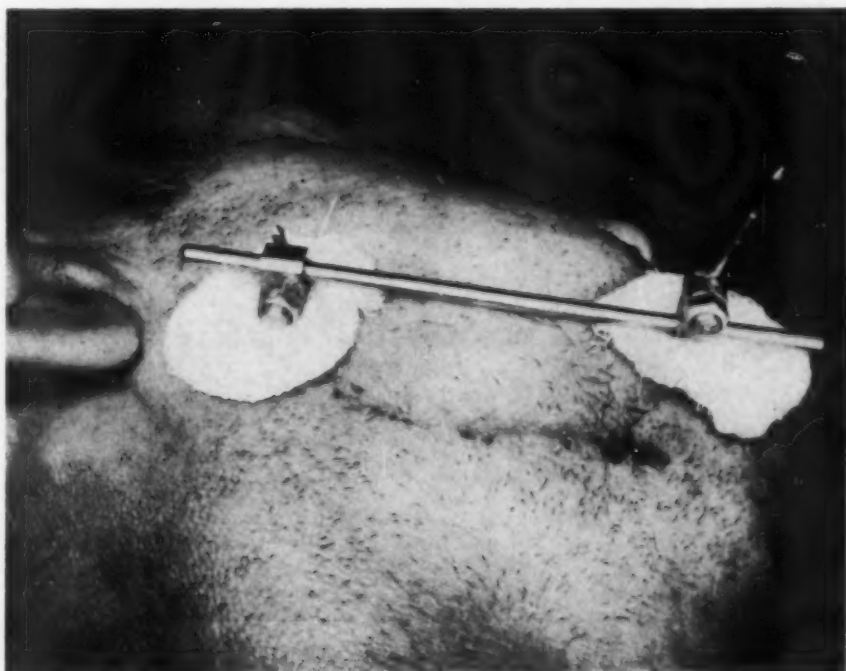


Fig. 489.—Frac-Sure bar attached to the pins to immobilize the fracture.

with a suitable instrument and taken out. Sulfanilamide powder, 1 Gm., was placed into each wound, which was closed in the usual way.

The postoperative x-ray taken after removal of the appliances showed a satisfactory result; the fracture appeared well united and the patient had good mobility of the jaw.

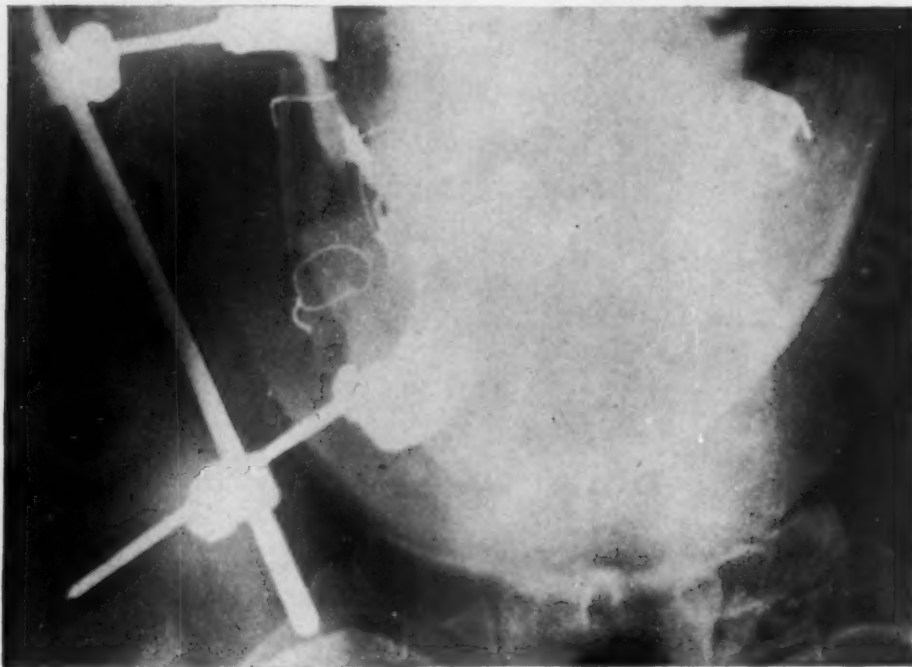


Fig. 490.—X-ray showing the bone graft in position, the fracture immobilized with peripheral bone clamp fixation, and the mandible by Jelenko splints.



Fig. 491.—Throat stick attached to the head to prevent the patient from rolling onto the operated side when sleeping.

Case 104

Oblique Fracture of the Mandible Treated by Direct Fixation With a Clamp

W. C. (416650), a 42-year-old man, came to the Emergency Ward on Jan. 22, 1945, with a swelling on the left side of the face and inability to open the mouth fully.

Four days before he had been struck on the left side of the face and had bled slightly from the mouth.

Examination showed swelling at the angle of the jaw on the left which was moderately tender to touch, and intraoral swelling around the lower left molar. The patient could not occlude the teeth, and the jaws would open only two-thirds of the way. The physical examination otherwise was negative except for chronic sinusitis.



Fig. 492.—X-ray showing fracture of the mandible involving the second molar.



Fig. 493.—Incision closed by interrupted sutures. The pin fixing the clamp extends through the stab incision.

X-ray examination showed a fracture of the left horizontal ramus of the mandible involving the second molar with only slight displacement of the fragments. There were two fracture lines, which is generally an indication of an oblique fracture (Fig. 492).

The patient was admitted to the House, and penicillin was administered intramuscularly, 96,000 units per day. The white blood count was 8,600. On January 26 under endotracheal gas, oxygen, and ether anesthesia the fracture was reduced. An incision was made below the inferior border of the mandible at the site of the fracture. The subcutaneous tissue and platysma were divided and the external maxillary artery and vein ligated and cut. The periosteum was incised and the fracture exposed. This fracture appeared to be oblique, overlapping about $\frac{3}{4}$ inch. A bone clamp was fitted directly over the fracture. The fragments were firmly fixed. The screw of the clamp extended to the outside of the skin through a stab incision; the pin was securely tightened. The subcutaneous tissue was closed by catgut suture and the skin by a subcuticular suture (Fig. 493). A small sponge was placed around the pin and covered with adhesive tape, and then a Barton bandage was applied to immobilize the mandible partially until the patient had recovered from the anesthesia.

The penicillin was continued for three days postoperatively. The patient made an uneventful recovery and was discharged on January 31 to be followed in the Outpatient Department.

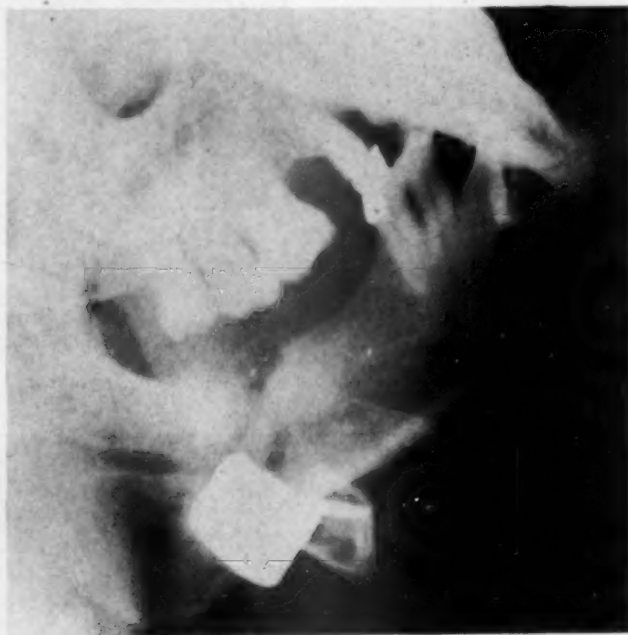


Fig. 494.—Postoperative x-ray showing the peripheral bone clamp holding the oblique fracture in position.

Postoperative x-rays showed the formerly reported fracture of the mandible, with the second molar removed and a clamp inserted at the inferior border directly over the fracture holding the fragments in good position (Fig. 494).

On February 2 the sutures were removed from the external wound and the dressings changed. On February 28 under novocain-epinephrine anesthesia, 4 c.c. injected locally, the scar on the left mandible was incised, and the incision carried through the subcutaneous tissue down to the clamp. The pin was removed, the clamp loosened and taken out. The wound was swabbed with zephiran and dusted with sulfanilamide powder. The musculature and the subcutaneous tissues were closed with catgut, and the skin with silk. The next day there was some swelling where the clamp had been removed. On March 5 the swelling had receded, and the patient was discharged on March 7, 1945.

MISCELLANEOUS CASE REPORTS

KURT H. THOMA, D.M.D., MARTIN WENIG, D.D.S., AND
SAMUEL I. KAPLAN, D.D.S.

CASES of special interest which were treated during the period covered by this issue included fractures of various types, infections of the jaws, odontogenic neck infections, various cysts, and tumors. A few of these will be reported.

Case 105

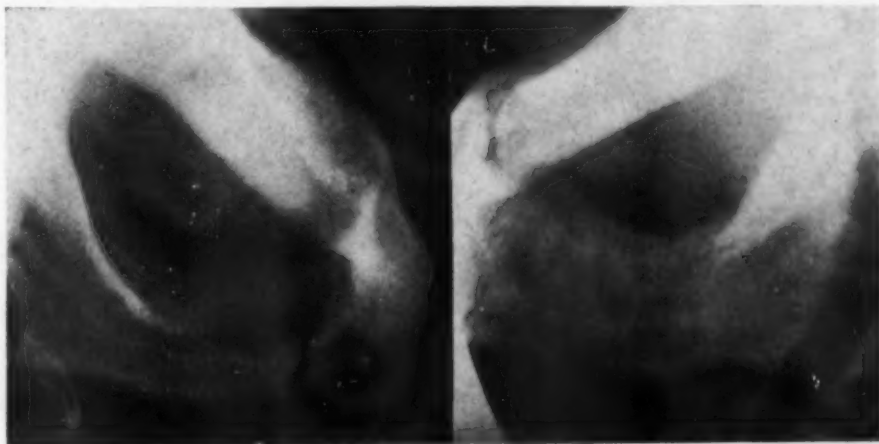
Bilateral Fractures of Mandible

G. M. (481346), a 43-year-old man, was brought to the Emergency Ward on March 6, 1945, suffering from cerebral concussion, fracture of the mandible, and lacerations of the ears.

The patient had been injured at work when a pile of heavy tank wheels fell on him. He was unconscious directly following the accident and was dazed for about one-half hour afterwards.

Examination revealed lacerations about the head and face, tenderness in the mandibular area, and discharge of blood from the ears.

X-ray examination showed no fracture of the skull, but bilateral fractures of the mandible at the level of the mandibular foramen (Figs. 495 and 496).



Figs. 495 and 496.—X-rays showing bilateral fractures of the mandible.

Diagnosis: Lacerations of the auricles and bilateral fractures of the mandible.

Plastic repair of the ears and lacerations was done on March 6. The patient received penicillin on the next four days, 32,000 units the first day, and 96,000 units the next three days. The mandible was temporarily immobilized by means of a head bandage and the two dentures inserted to act as splints.

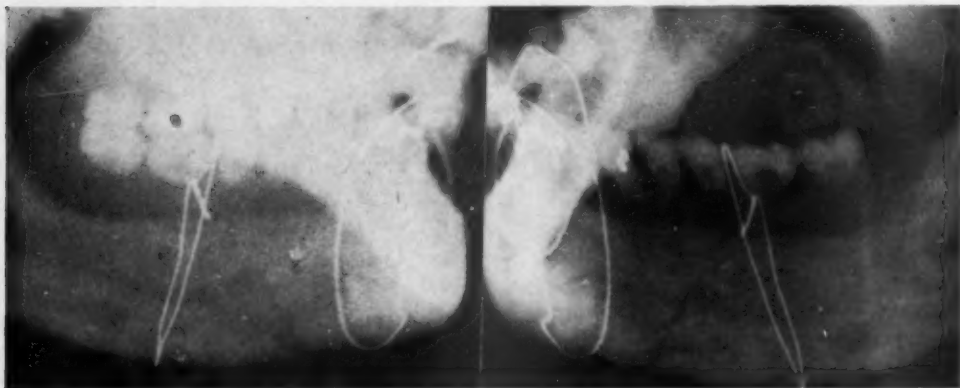
On March 13 reduction of the bilateral fractures was performed with circumferential wiring of the jaw. Under endotracheal gas, oxygen, and ether anesthesia, after the usual preparation of the mouth and skin, the sterilized lower denture was inserted on the alveolar ridge and the fractured jaw reduced by molding it into the denture. Circumferential wires were then inserted by means of the hypodermic needle technique, placing a 25 gauge stainless steel wire around the mandible and over the denture in the anterior region and posterior to the fractures on each side of the mandible. These wires were then tightened so that the mandible was properly splinted. The upper denture was inserted and the occlusion was found

to be normal. A Barton bandage was applied. The patient left the operating room in good condition.

The patient received sulfadiazine therapy on the day before reduction of the jaw, 2 Gm. to start and 1 Gm. every four hours; this was continued for the next three days.

Postoperative x-rays showed three wire loops holding the fragments in position with the lower denture used as a splint. Good position was maintained (Figs. 497 and 498).

The patient was discharged on March 18 to be followed at the office. He was seen biweekly for cleansing the mouth with a spray and checking on the progress of healing. The aftercare was uneventful and on April 18 the wires were removed. The patient had slight muscular trismus, but the fracture had united in good position. After one week the patient reported that he had regained normal range of mobility of the jaw.



Figs. 497 and 498.—Postoperative x-rays showing peripheral wire fixation with the lower denture used as a splint.



Fig. 499.—Photograph showing swelling on the side of the maxilla.

Case 106

Maxillary Cyst

E. E. (478361), a 34-year-old man, came to the Dental Clinic on Jan. 31, 1945, complaining of a swelling on the left upper maxilla.

About two years before the patient had had a tooth extracted in the upper left second premolar region. After this his "gum swelled up." A local dentist incised it, and the swelling went down only to return again. The patient was referred to the hospital.

Examination showed a swelling on the side of the upper jaw (Fig. 499). X-ray examination showed a cystlike defect in the upper jaw in the premolar area. There was no evidence of retained roots in this area. The root of the upper left canine seemed to be very close to the defect and possibly was connected with it. The sinuses appeared clear.

Diagnosis: Cyst or tumor.



Fig. 500.—Photograph showing cyst being enucleated.



Fig. 501.—Incision closed by sutures.

The patient was admitted to the hospital on March 16, 1945, and on the next day excision of the cyst was performed. Under intravenous pentothal sodium anesthesia, after the usual preparation of the face and mouth with zephiran, a U-shaped incision was made around the bulging tumor on the outside of the maxilla on the left. The mucoperiosteum was dissected away from the bone which was found to be bulging and thin. It was of a greenish appearance,

which seemed consistent with that of a cyst. After the outer bony wall was removed, the cyst was detached from its internal bony wall with a periosteal elevator, and a sac about 3 cm. in diameter was removed intact (Fig. 500). It seemed to be filled with a fluid. The hemorrhage was stopped with adrenalin so that the bony compartment could be inspected. It was found that a perforation existed in the internal posterior aspect of the cyst cavity, which seemed to enter the maxillary sinus. The partition between the cyst and sinus was removed so that the cyst cavity and maxillary sinus were made into one. With a trocar and rasps an opening was made in the nasointral wall; a petrolatum strip was inserted from the cavity into the nose and allowed to project through the nostril. The remainder of the strip was packed into the combined cyst and sinus cavity. Then the flap was returned and sutured into position (Fig. 501). A piece of adhesive tape was used to attach the end of the strip projecting from the nose to the cheek.

The patient received sulfadiazine therapy, 3 Gm. to start and 1 Gm. every four hours for four days postoperatively. On the third postoperative day the pack was removed through the nostril, and the sutures in the mucosa removed. The incision was irrigated with saline and painted with methylene blue. The patient was discharged on March 23 to be followed at the office.

Pathologic report, gross: A soft fluctuant cyst measuring 3 cm. in diameter; the outer surface was reddened and covered by a few fibrous tags. It was filled with turbid brown fluid. The inner lining was reddened and slightly roughened. The cyst was lined by squamous epithelium. There was chronic inflammation present. Diagnosis: Cyst.

Case 107

Globulomaxillary Cyst

M. DeC. (494561), an 18-year-old girl, presented a swelling in the left maxillary canine region with a peculiar xanthomatous spot (Fig. 502).

About two months ago the patient noticed a swelling on the left buccal gingiva with a small discharging fistula.



Fig. 502.—Photograph showing swelling of the maxilla with xanthomatous area.

X-ray examination showed a lateral deviation of the roots of the canine and second incisor tooth. From between these roots a cystic area extended up from the alveolar process into the maxillary region. None of the teeth were carious or appeared infected. The incisor which seemed to be projecting into the cystic area was normal except for a small filling.

Diagnosis: Globulomaxillary type of fissural cyst (Fig. 503).

The patient was admitted to the hospital on June 25, 1945, and the next day under gas, oxygen, and ether anesthesia the cyst was excised. A horizontal incision was made on the right upper maxilla beneath the lip. It was about 2 inches in length and extended

close to the gingival margin. A vertical extension was made on each end. The mucoperiosteal flap was dissected away from the very thin bulging bone (Fig. 504) into which a window could be cut with a knife. The cyst was then visible. The cyst cavity contained a thin epitheliated sac which was detached and removed in one piece. Inspection of the wound showed that none of the teeth was involved (Fig. 505). The roots were merely forced apart by the cystic pressure. A boric strip was inserted and the flap pressed into the bottom of the cyst cavity.



Fig. 503.—X-ray of maxilla showing globulomaxillary cyst.



Fig. 504.



Fig. 505.

Fig. 504.—Mucoperiosteal flap dissected away from the bulging bone.

Fig. 505.—Bone cavity after removal of the cyst sac.

The patient made an uneventful recovery and was discharged on June 29 to be followed at the office. She was seen several times to have new dressings applied. These were discontinued on July 5, and she was instructed to irrigate the wound with a soft rubber ear syringe.

Pathologic report: A cyst measuring 1.5 cm., previously opened.

Diagnosis: Cyst with acute and chronic inflammation.

Case 108

Dentigerous Cyst of Mandible

H. D. (202479), an 8-year-old boy, was referred to the Dental Clinic on April 25, 1945, with a diagnosis of cyst of the mandible.

A few days previously his local doctor had noticed what appeared to be an alveolar abscess of the mandible. The lower right deciduous canine was removed, and because of the nature of the discharge, the presence of a cyst was suspected. This was confirmed by x-ray examination and the patient was referred to this hospital.

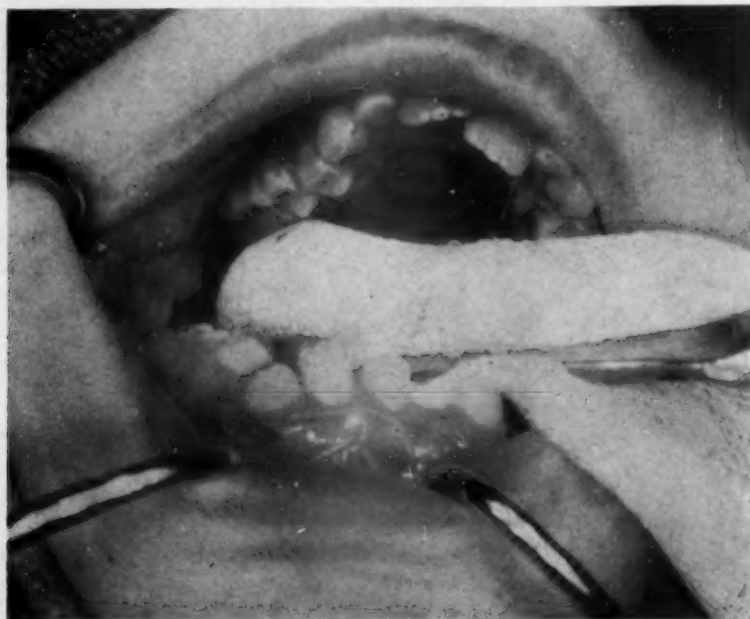


Fig. 506.—Fluctuant swelling on the side of the mandible causing malocclusion.



Fig. 507.—X-ray showing dentigerous cyst. Note displacement of the developing premolars and canine.

Examination showed a hydrocephalic boy in no acute distress with a small inguinal hernia on the right. There was a fluctuant swelling on the buccal mucosa of the mandible in the lower right premolar region (Fig. 506).

X-ray examination revealed a dentigerous cyst in the lower right mandible containing the lower right permanent canine and first premolar tooth (Fig. 507).

The patient was admitted to the House on April 27, 1945. Blood examination showed a white cell count of 11,000; red cell count, 4,500,000; and hemoglobin 13 Gm. Three days after admission an operation for removal of the cyst was performed. Under endotracheal gas, oxygen, and ether anesthesia, an incision was made on the alveolar crest of the lower right premolar area of the mandible, with vertical extensions from each end. A U-shaped mucoperiosteal flap was dissected away from the wall of the cyst and retracted. The remaining portion of the cyst was carefully dissected away from the bone (Fig. 508). The unerupted

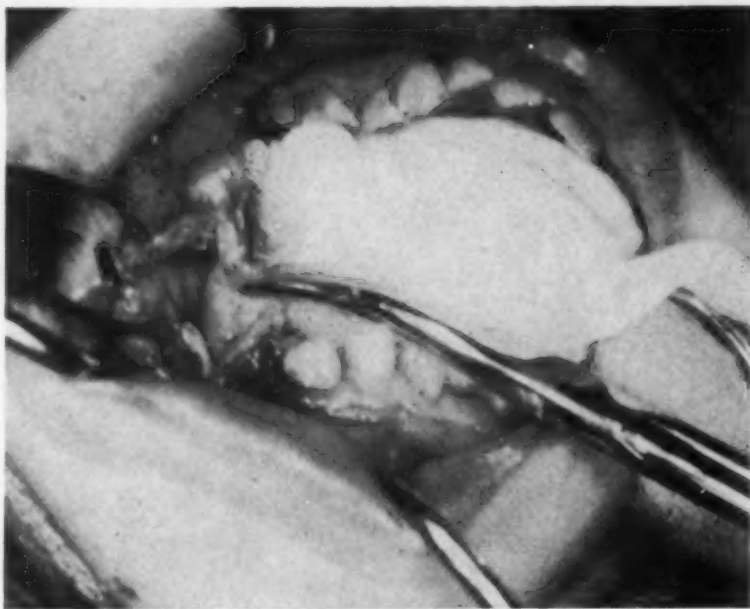


Fig. 508.—Removal of cyst sac.

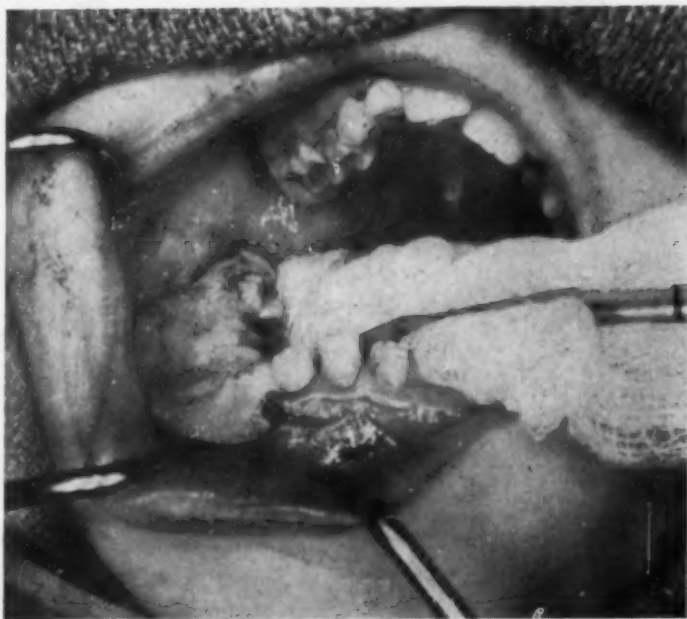


Fig. 509.—Borated gauze strip inserted into wound.

permanent canine and first premolar were removed. It was decided to try and save the developing second premolar which was exposed by the dissection of the cyst, and did not appear to be included in it. Two grams of sulfanilamide powder were inserted into the wound. The mucoperiosteal flap was folded into the cavity which was then obliterated with a borated gauze strip (Fig. 509). Interrupted dermalon sutures closed the vertical incisions.

The day following the operation the patient's temperature rose to 102.4° F. He was given penicillin, 16,000 units every three hours for three days. During this time the temperature dropped to normal. The patient was discharged on May 5, 1945, to be followed in the Outpatient Department. Dressings were changed every three days, the wound irrigated with Dobell's solution, and a new borated dressing inserted until the entire bone cavity was covered with granulation tissue. He was then instructed to clean the cavity by irrigation until it had become completely obliterated.

Pathologic examination: The specimen consisted of an oval cystic structure measuring 3.5 by 1.5 by 1.5 cm. (Fig. 510). **Diagnosis:** Dentigerous cyst.



Fig. 510.—Photograph of cyst sac containing an unerupted canine (C) and a tooth follicle with premolar (P).



Fig. 511.—X-ray showing small cystic area containing recurrent adamantoblastoma.

Case 109

Recurrent Adamantoblastoma

H. C. (420291), a 49-year-old man, was admitted to the hospital on Feb. 5, 1945, for excision of a recurrent adamantoblastoma. He had been operated on, Sept. 10, 1943; his past history and the first operation are described in *Volume II*, Case 44. He was periodically examined for recurrence, but as he lived out of town he finally omitted the check-up examinations. He was not seen between Dec. 1, 1943, and Dec. 1, 1944. At this time a hard nodular swelling was present on the labial surface of the excision area.

X-ray examination showed a small cyst extending from the alveolar border on the labial side of the ridge in an edentulous area, evidently the site of the first incision (Fig. 511).

Chemotherapy was started on February 6, 2 Gm. sulfadiazine to start, and 1 Gm. every six hours, because the patient had a cough and inflamed throat and tonsils, and the operation was postponed a few days.

On February 8 under endotracheal gas, oxygen, and ether anesthesia, the tumor was excised. Four cubic centimeters of monacaine-epinephrine were injected both on the outside and the inside of the anterior part of the mandible. Two teeth were extracted—the left lower second premolar and the right lower incisor. An incision was made in the gingiva

around the tumor mass, and about $\frac{1}{2}$ cm. away from it on both the labial and lingual sides. The mucoperiosteum was elevated to expose the unaffected part of the mandible. By means of a drill a horizontal incision was outlined in the bone, about $1\frac{1}{2}$ cm. below the tumor. With a circular saw the bone was cut vertically at each side so as to outline a block $1\frac{1}{2}$ inches in width and 1 inch high containing the tumor mass to be excised. By means of an osteotome the block was completely detached, after the genioglossus muscle was cut from the mandibular spine. The edges of the bone were smoothed and débrided, and the bone carefully inspected. No evidence of tumor tissue was found. A boric strip was placed on the osteotomized surface of the bone and stay sutures were placed between the labial and lingual incisions.

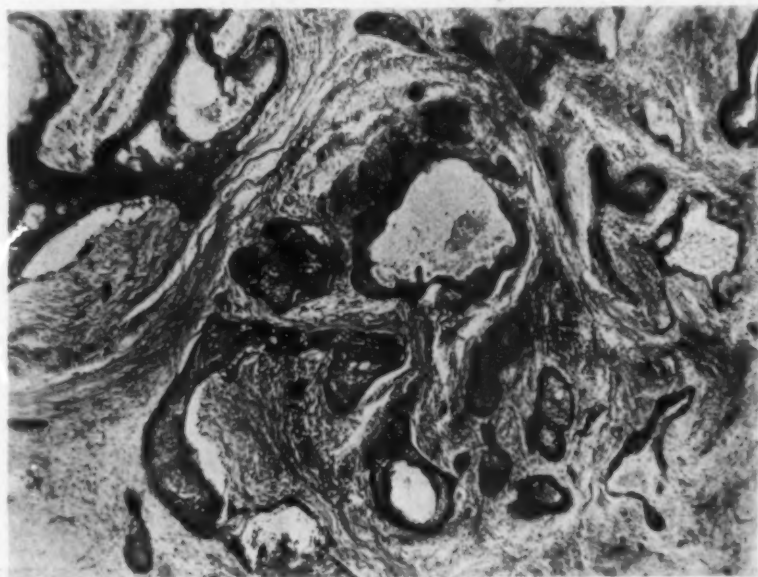


Fig. 512.—Photomicrograph of adamantoblastoma invading the marrow spaces.

The sulfadiazine was continued for four days postoperatively. The patient made an uneventful recovery and was discharged on Feb. 14, 1945.

Pathologic examination of a sagittal section through the excised bone of the mandible showed epithelial cells forming follicles and invading the marrow spaces (Fig. 512). Diagnosis: Adamantoblastoma.

Case 110

Odontoma

S. R. (478101), an 8-year-old boy, was referred to me because the left upper deciduous incisor failed to be shed.

Six years ago this incisor failed to erupt normally. At that time a small cystic condition was discovered (Fig. 513); this was not removed.

Examination showed a well-developed boy with a diastema in the median line and a hypertrophied labial frenum. On the right side the two permanent upper incisors were present; on the left the two deciduous ones had been retained.

X-ray examination revealed a calcified mass which was identified as irregularly formed miniature teeth (Fig. 514). Distal to this mass were three unerupted teeth which, in other x-ray views, could be identified as the permanent first and second incisors and canine. An odontoma had formed, not from a regular tooth germ, but in the cystic area discovered seven years ago. This must have contained a soft odontoma which later expanded and formed calcified structures.

The patient was admitted to the hospital on Jan. 31, 1945, and on the next day the tumor was excised under gas, oxygen, and ether anesthesia. An incision was made on each side of the frenum, both labially and palatally. It was grasped with a hemostat and after a horizontal incision at the base of the lip was made, it was completely excised. An incision was then made on each side of the left first and second incisors, and the two teeth extracted.

The incision was then extended on each end both on the labial and palatal sides, and a muco-periosteal flap retracted. The alveolar bone was then removed by means of a chisel until the hard tumor mass was exposed. It consisted of two main portions each made up of a number of small rudimentary teeth held together by osseous structure. After these masses were removed a clean bone cavity was found to extend up into the region where the permanent teeth generally form. By removing more bone on the distal side of this cavity, the permanent



Fig. 513.—Small cystic area present when patient was 2 years old.

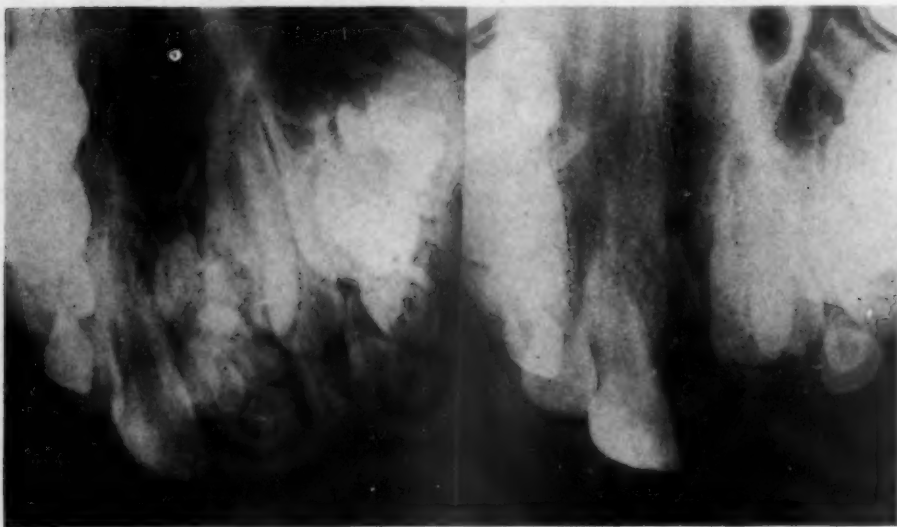


Fig. 514.

Fig. 515.

Fig. 514.—X-ray showing odontoma which has developed in the cystic area.
Fig. 515.—Postoperative x-ray after removal of odontoma.

first incisor was found, and its crown partly exposed and freed from osseous structure to facilitate eruption (Fig. 515). The permanent second incisor could not be demonstrated. It was probably located palatally, and it was felt that the eruptive force of the tooth would bring it to the surface later. The incisions were closed by means of Kaldermic interrupted sutures.

Recovery was uneventful and the patient was discharged Feb. 2, 1945.

Case 111

Central Giant-Cell Tumor

F. S. (489458), a 64-year-old woman, was admitted on May 10, 1945, for excision of a central giant-cell tumor of the mandible.

Seven years before the teeth in the anterior part of the mandible had become sore and the gingiva had begun to swell. X-ray examination showed a cyst with an indefinite margin (Fig. 516). This was found to be a central giant-cell tumor. The teeth from the right lower canine to the right lower first incisor were extracted, and the tumor excised.

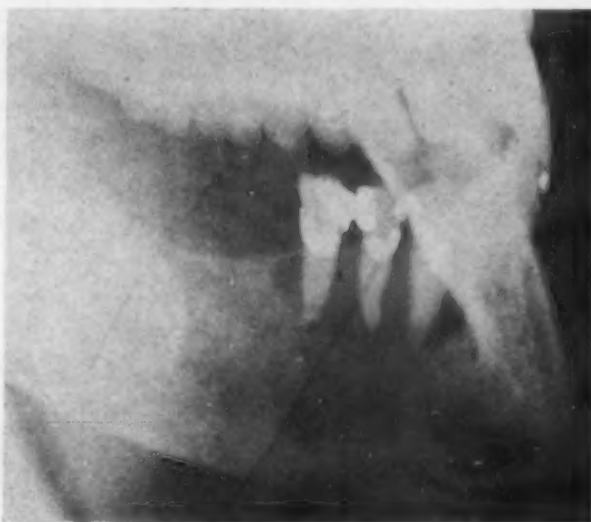


Fig. 516.—X-ray taken six years before when patient had a giant-cell tumor on the left side of the mandible.

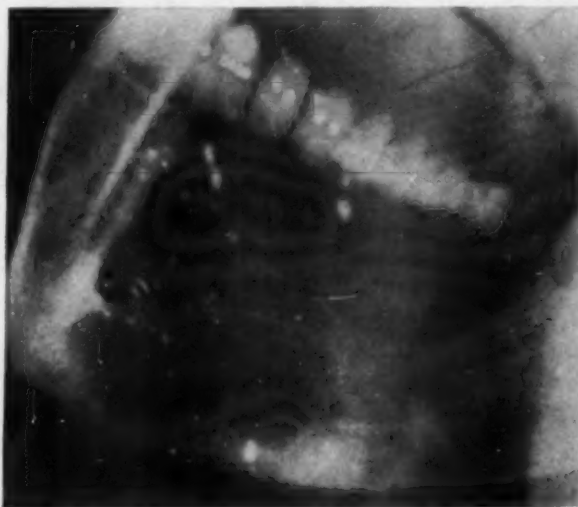


Fig. 517.—X-ray showing a new giant-cell tumor which had developed on the right side of the mandible.

About one year before a swelling started to form on the right mandible which increased slowly in size until the patient could no longer wear her dentures. Examination showed a painless tumor mass the size of a goose egg expanding the left mandible.

X-ray examination showed an osteolytic defect in the bone of the left side of the mandible measuring about 4 cm. in greatest length. The margins were irregular and seemed to be broken through the alveolar ridge. Numerous areas of increased density were scattered throughout the soft tissues of the floor of the mouth, probably old lipiodol which had been used to make a differential diagnosis (Fig. 517).

Diagnosis: Central giant-cell tumor.

On May 11, the day after admission, the blood calcium was 12.2 mg. per cent, the phosphorus, 1.7 mg. per cent, and phosphatase, 3.4 units. Under intratracheal gas, oxygen, and ether anesthesia, the mouth was prepared in the usual manner and 4 c.c. monacaine-epinephrine injected below the mucosa in the area of the tumor. An incision was made on the alveolar ridge, extending over the entire tumor surface. The mucosa was carefully dissected away from the expanded mandible, and the thin shell of bone was incised by means of an osteotome in the place where the bone was solid. By means of a large curette most of the tumor mass was enucleated and removed with the shell of bone. There was, however, a great deal of oozing brownish tissue remaining, and this was thoroughly curetted out. Several small chambers were found and cleansed in this manner. On the posterior end of the tumor cavity the tumor was found to extend into the mandibular canal. This area was thoroughly curetted and opened wide. The mandibular nerve was seen and preserved. The ensuing hemorrhages, particularly that of the mandibular artery, were arrested with fibrin foam and thrombin. The cavity was inspected and all tumor tissue was found to have been removed. It was then treated with 95 per cent phenol which was neutralized with 95 per cent alcohol, followed by Hexyl-Resoreinol Solution S.T. 37. The fibrin foam which had taken up some of the phenol was removed and replaced although the hemorrhages had completely stopped. A boric gauze pack was inserted into the wound, and a Barton bandage applied. The patient's blood pressure dropped somewhat during the operation and she was given an intravenous infusion of 1,500 c.c. of 5 per cent dextrose in water.

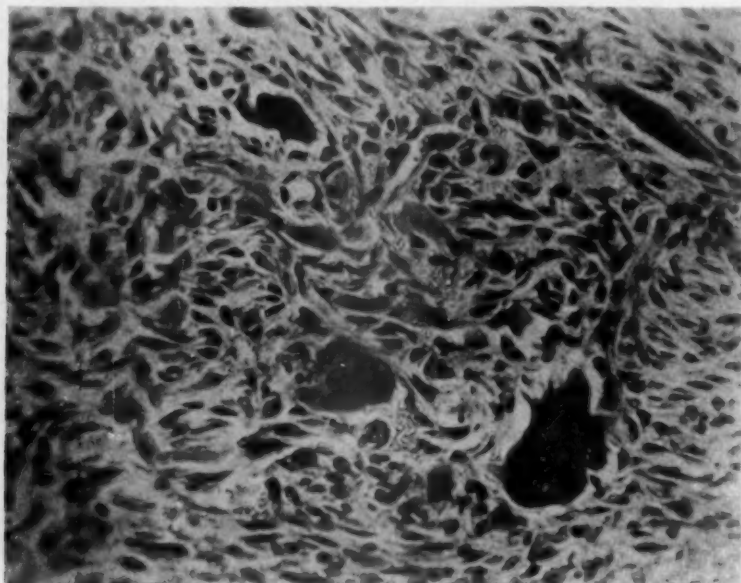


Fig. 518.—Photomicrograph of giant-cell tumor.

The patient made an uneventful recovery and was discharged on May 14, 1945, to be followed at the office. She reported for dressings until the wound had completely healed. She was discharged on July 5, 1945, to be re-examined at intervals of three months for recurrence.

Pathologic examination: The specimen consisted of several pieces of reddish-brown tissue containing bony material. Diagnosis: Benign giant-cell tumor (Fig. 518).

Case 112

Leucoplakia and Papilloma of Tongue

M. B. (483656), a 60-year-old woman, came to the Dental Clinic on March 26, 1945, complaining of a "sore tongue." She also had a lump on the tongue. Six months before the patient noticed a "small pimple" on the surface of the tongue. While scraping it with a knife she cut the pimple and "drew blood." For the past two months the tongue had been painful, waking her at night, but for the past two weeks, she had been comfortable. She had lost a few pounds of weight in the last six months; otherwise her general health had been good.

Examination of the tongue revealed an area of leucoplakia with a small papillomatous lesion which apparently had not extended deeply into the tongue. There was no ulceration (Fig. 519). Physical examination showed essential hypertension with a question of hypertensive heart disease with annular fibrillation. Blood examination on March 26 showed the hemoglobin to be 14.3 Gm., and the red cell count 4,510,000.

Diagnosis: Papilloma and leucoplakia of tongue.

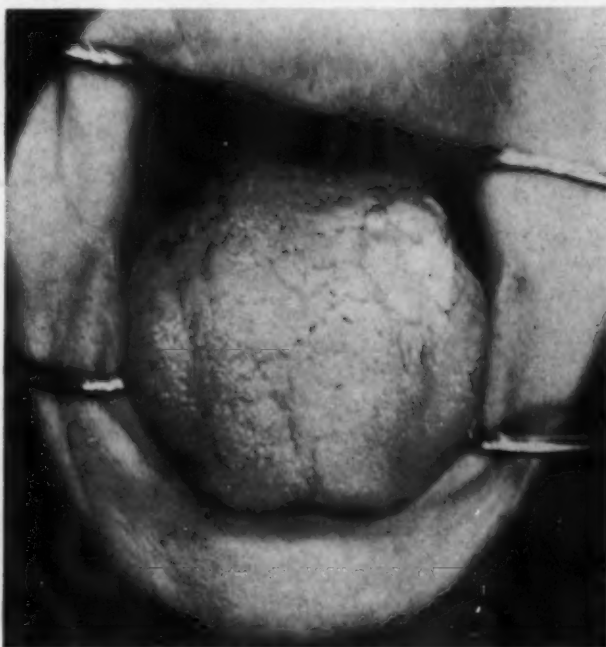


Fig. 519.—Leucoplakia of the tongue with papillomatous formation.

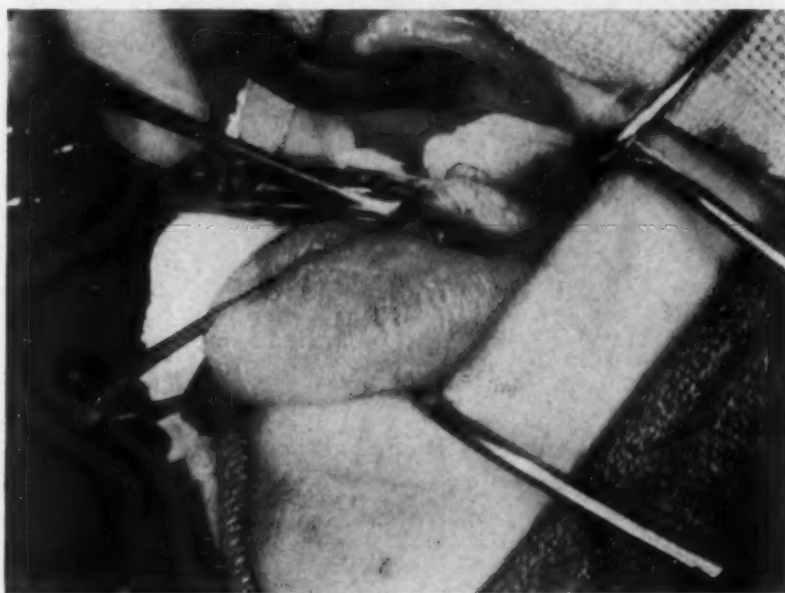


Fig. 520.—Excision of papilloma of the tongue.

On March 27 the patient was seen in Tumor Clinic Conference, and it was the feeling of the group that the local lesion should be completely excised and that, if malignant, a hemiglossectomy should be done. A neck dissection should follow if such were the case, as many times there are multiple lesions deeper in the tongue.

The patient was admitted to the House on March 27 for excision of the papilloma. The next day, under endotracheal gas, oxygen, and ether anesthesia, and after preparation

of the skin and mouth with zephiran, 6 c.c. of monocaine were injected below the tumor into the tongue to arrest capillary bleeding. Tongue forceps were inserted in the median line in order to pull the tongue forward. An elliptical incision was then made, including not only the papilloma but also the area of leucoplakia (Fig. 520), removing about $\frac{3}{4}$ inch of the muscle layer beneath the surface with the specimen (Fig. 521). After several bleeding vessels were tied, the incision was closed by vertical mattress sutures and interrupted dermalon sutures (Fig. 522).



Fig. 521.—Wound after excision.



Fig. 522.—Wound closed by sutures.

On the second postoperative day the tongue became swollen and ecchymotic. An ice collar was applied, and penicillin therapy instituted; 48,000 units were given on the first day and 96,000 units intramuscularly on the next three days. Hydrogen peroxide mouth-washes were used after each meal. The patient improved and was discharged on April 3 to be followed in the Outpatient Department.

One week later the wound was well healed, and the sutures removed. There was still evidence of atrophy of the filiform papillae on both sides. Vitamin B complex was prescribed, and the patient discharged to return in two months for a check-up.

Pathologic examination: An oval piece of muscle covered by bluish-gray epithelium, measuring 2.5 by 1.5 by 1 cm. In the center of the epithelial surface there was a reddish raised papillary mass measuring 7.5 mm. in diameter. Diagnosis: Papilloma (Fig. 523).



Fig. 523.—Photomicrograph of papilloma of the tongue.



Fig. 524.—Photograph of carcinoma of the tongue.

Case 113

Carcinoma of the Tongue

M. G. (484830), a 57-year-old woman, was admitted to the hospital on April 2, 1945, with a tumor of the tongue. About four weeks previously the patient had noticed a lesion on the anterior right border of the tongue. It gradually increased in size, and became irritated by the lower teeth.

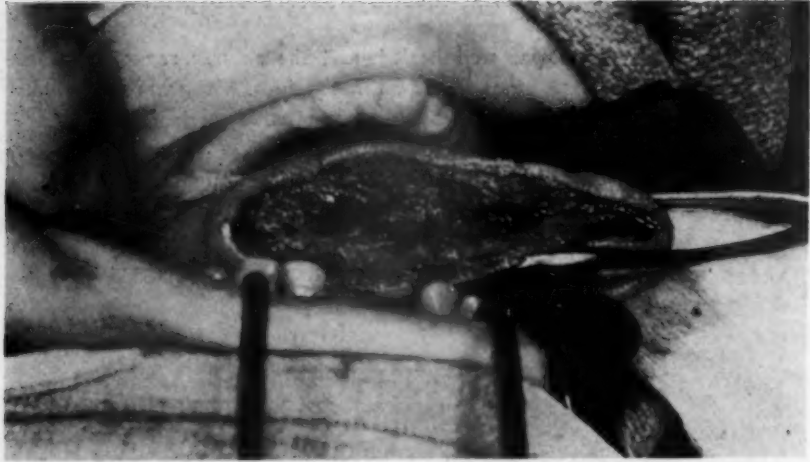


Fig. 525.—Wound after excision of carcinoma.

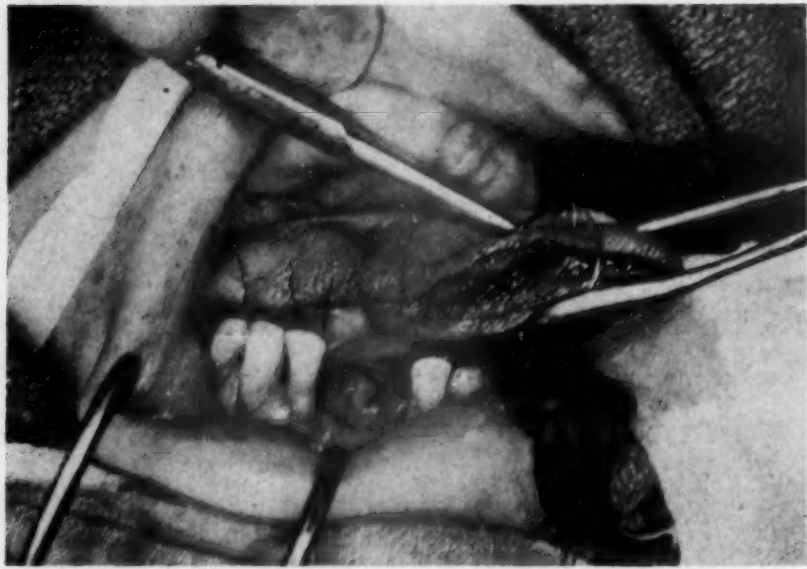


Fig. 526.

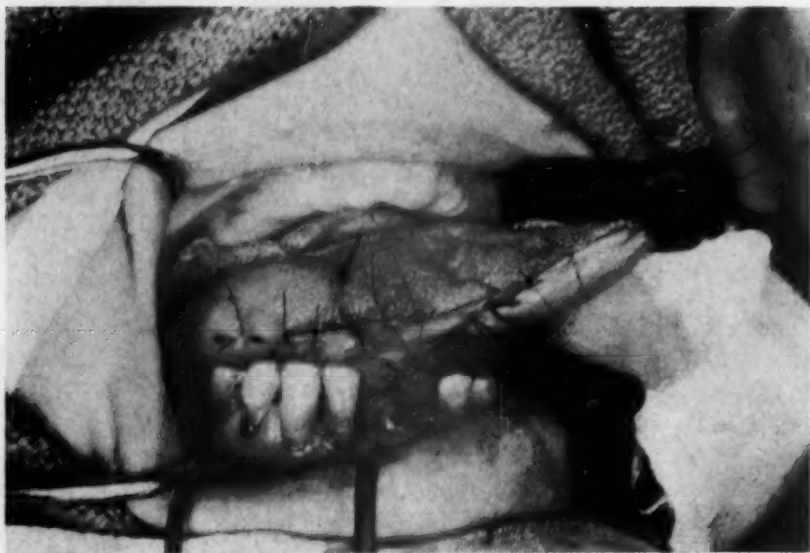


Fig. 527.

Figs. 526 and 527.—Closing the wound with interrupted sutures.

Examination showed a lesion $2\frac{1}{2}$ by $1\frac{1}{2}$ cm. on the right anterior border of the tongue; it was ulcerated in the center with indurated edges (Fig. 524). The tongue was also tied by a fibrous band of tissue and could not be protruded. There was a grade II systolic murmur of the heart, maximal at the aortic area. Diagnosis: Tongue-tie and carcinoma.

On April 3 excision of the carcinoma was performed and the tongue-tie corrected. Under gas, oxygen, and ether anesthesia, the face and mouth were prepared in the usual manner with zephiran. Tongue forceps were inserted into the tip of the tongue. An incision was made in the heavy strand of tissue which represented the frenum and tied down the tongue so it could not be protruded. The ensuing gap was closed with ten interrupted silk sutures taken in the floor of the mouth and the undersurface of the tongue.

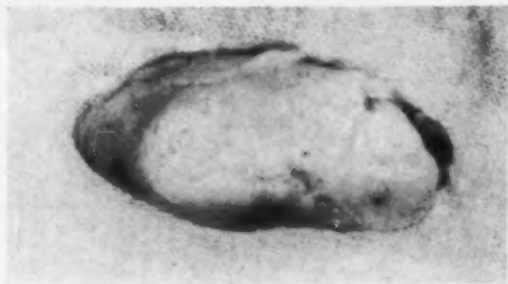


Fig. 528.—Photograph of gross specimen of carcinoma.

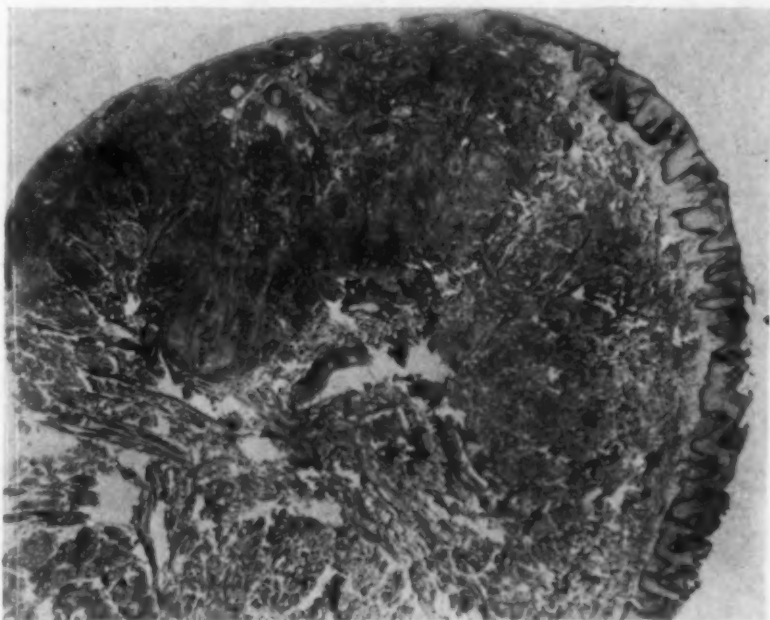


Fig. 529.—Photomicrograph of section of carcinoma of the tongue.

Excision of the carcinoma was next performed. The tongue forceps were inserted further back and a suture taken in the posterior part of the tongue with heavy silk for traction. The tongue was protruded. Monocaine-epinephrine, 3 c.c., was injected for hemostasis, and the lesion was excised with a margin of about 15 mm. (Fig. 525). The mucosa both on the dorsum and on the underside was undermined by cutting the section out in the form of a V to allow better closure of the wound. After the section was removed, blood vessels were tied and two vertical mattress sutures inserted to approximate the margins of the wound. The incision was then closed by interrupted dermalon sutures (Figs. 526 and 527). The patient was discharged from the operating room in good condition.

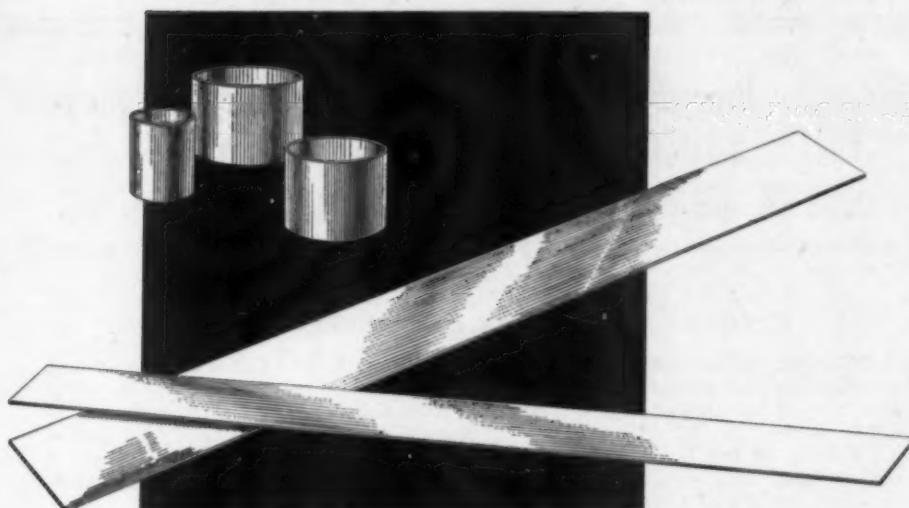
The patient received an intravenous infusion of 1,500 c.c. dextrose in water after the operation. The wound was painted with 2 per cent methylene blue the next day, and the patient was given hydrogen peroxide mouthwashes after every meal. She made an uneventful

recovery and was discharged on April 8, 1945, to be followed at the office for periodic check-up examinations.

Pathologic report: A piece of tissue measuring 3.5 by 2 by 1.5 cm. and covered on one aspect by mucous membrane was submitted for examination (Fig. 528). On the membranous aspect there was an ulcerated area measuring 1.5 cm. in diameter surrounded by induration. On section this area was grayish-white, firm, and granular. Diagnosis: Epidermoid carcinoma, grade I (Fig. 529).



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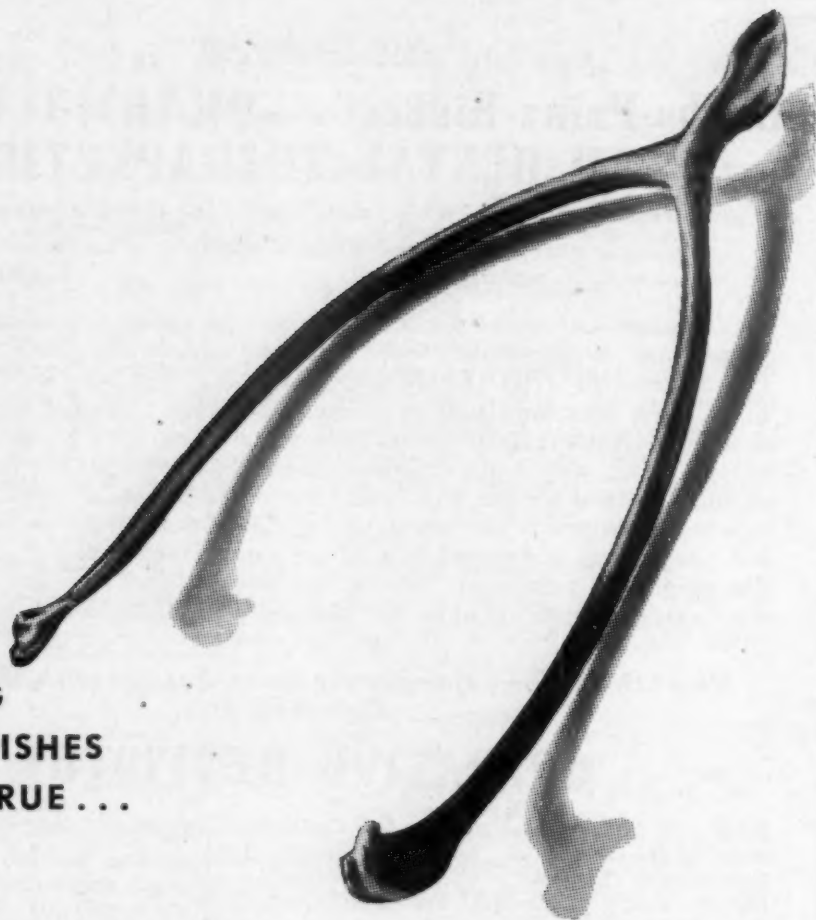
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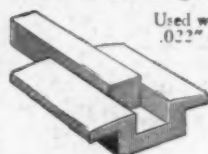
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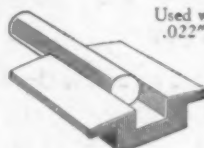
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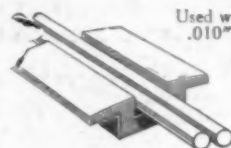
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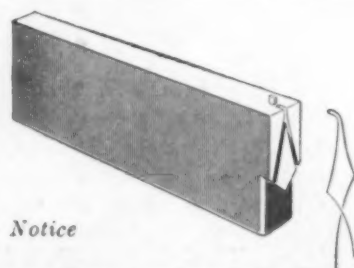
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Volume II

ALBERS-SCHÖNBERG DISEASE

REPORT OF TWO CASES

GORDON R. WINTER, D.D.S., F.A.C.D.*

ALBERS-SCHÖNBERG disease is a condition characterized by extraordinary thickness and density of the cortex of the bone which encroaches on the medullary portion. In many instances the spongiosa is completely obliterated.

This disease is also known as: osteopetrosis, osteocretosis, congenital osteosclerosis, osteosclerosis fragilis generalisata, disseminated condensing osteopathy, marble-bone disease, and chalky-bone disease. Heinrich Albers-Schönberg, a German radiologist, in 1904, was the first to recognize this bone dysplasia. In 1926, Karshner suggested the term, "osteopetrosis," which means "stone-like bones" and, in 1937, Smith originated the name, "osteocretosis," meaning "chalklike consistency of bones."

During the past forty years, approximately 135 cases of this disease have been recorded, but, in the earlier literature, many cases reported as osteosclerosis with leucemia may have been osteopetrosis. A few instances of this condition have been recognized in utero while some have not been identified until the sixty-eighth year of life. They are about equally divided between males and females, and, in a striking percentage, consanguinity has been present.

Albers-Schönberg disease may be suspected clinically by its secondary manifestations, the most important of which are:

Multiple Fractures.—These fractures generally occur at right angles to the shafts of the long bones and heal in a normal length of time. Spontaneous fractures, which have healed and which the patient has never been aware of, are sometimes recognized in radiographs.

Optic Atrophy.—The atrophy of the optic nerve is caused by narrowing of the optic foramina. The bony impingement and thickening of the base of the skull readily account for this condition. Complete blindness is often an end result.

Blood Dyscrasias.—Various blood dyscrasias are frequently found. Anemia, varying from a mild form to a severe aplastic anemia—the result of mechanical replacement of the bone marrow—is a common terminal manifestation. Whenever leucemia is noted, it is usually of the myelogenous type.

Adenopathy, Hepatomegalia, Splenomegalia.—The enlargement of the liver, spleen, and lymph nodes may be a compensatory mechanism following hypoplasia of the bone marrow. Some histologic studies of these structures have shown little increase in the blood-forming tissues but an increase in the connective

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tive tissue. A few writers have stated that osteopetrosis is a disease of the entire hematopoietic system and not a disease of the skeleton with secondary changes in the blood-forming organs. It is well to remember that, at an early period in embryonic life, hematopoiesis becomes localized in the liver, spleen, bone marrow, and lymph nodes. Later in prenatal life the liver and spleen lose this function and the bone marrow and lymphoid tissue carry on.

Albers-Schönberg disease has been divided into two types—malignant and benign. The malignant type is identified early in life and presents the condition in its severest form with marked marbling of the bones, multiple fractures, anemia, leucemia, optic atrophy, and enlargement of the liver, spleen, and lymph nodes. The appearance of the patient is strikingly characteristic. The face, which is broad, has a senile expression. The nose is flat with the nares widely separated, the lips are thickened, eyes far apart, bosses prominent, hair abundant, and skin wrinkled. Other conditions are usually present, such as: hydrocephalus, nystagmus, deafness, osteomyelitis, mental deterioration, delayed physical development, retarded dentition, and early dental caries. The life span is short. Death is usually caused by an intercurrent infection or by some type of blood dyscrasia.

The benign type, as the name implies, is not fatal. The condition develops slowly and is sometimes not recognized until late maturity. Children and adults are known to have the disease without apparent symptoms, the diagnosis being made incidental to some concomitant condition. In fact, it is often discovered coincidentally with a radiographic study of some part of the body. The various secondary manifestations are usually absent, or present in a mild form. Furthermore, the age of the patient bears no relationship to the amount of marbling, as some children reveal more than some adults. A few commentators feel that sclerosis is more progressive during the period of growth when the epiphyses are still open, but this does not appear to have been substantiated. The progress of the disease apparently varies with individuals.

The most striking variations in Albers-Schönberg disease are found in the osseous tissues, which are altered physically though but slightly chemically. The substantia spongiosa, which is encroached upon by the cortex, is frequently changed by the number and thickness of the individual trabeculae. The resulting density is grossly indistinguishable from the compacta as shown in biopsy. This alteration takes place by endosteal and endochondral bone metabolism, the periosteum remaining more or less normal. Histologically, the bone is disorderly and irregular in pattern, with reduction or obliteration of the intertrabecular spaces, marrow cavity, and Haversian canals.

Opinions differ as to the manner of production of these bony defects. Some authors report evidence of abnormal osteoblastic activity, and others feel that it is unusual osteoclastic action. A few believe that the narrowing of the medullary canal and its apparent obliteration are not real. This conclusion is the result of studying radiographs of the skeletal structure alone, especially in cases where an autopsy has not been performed. These investigators think that the abnormal radiographic picture is caused by excessive calcareous deposits in the cortex, thereby preventing the demonstration of the medullary canal. This is advanced as an explanation for the absence of anemia and the lack of enlargement of the liver, spleen, and lymph nodes in some cases. Throughout the literature, patients have been recorded with almost complete obliteration of the marrow cavity, as seen radiographically, yet presenting an essentially normal blood picture.

It is evident from the biopsies and post-mortem examinations reported in the published data that there are two types of bone, one that is difficult to cut and one that yields readily to instrumentation. This, no doubt, accounts for the absence or prevalence of fractures. The tendency for the bones to fracture rather than to bend in this disease is brought about by the loss of elasticity. This brittle condition resembles the bones of senility where there is an increase in the mineral content.

Other characteristic bone changes have been noted in certain areas. In the sella turcica clubbing of the posterior clinoid processes is sometimes observed. The long bones often present alternate transverse bands of extremely dense and more nearly normal bone parallel to the epiphyseal lines. Clubbing of long bones is also frequently noted.

Some investigators feel that the salient features of the chemical analysis of marble bones are the high carbonate content and hypermineralization, while others claim no change in the calcium, phosphorus, magnesium, or carbonate content. One writer states that a disturbed calcium metabolism characterized by augmented ability to bind calcium exists; the nature of this process is not clearly understood. In the blood serum, most investigators believe that the calcium and phosphorus are within normal limits but, again, a few have found the blood phosphorus to be increased.

DIFFERENTIAL DIAGNOSIS

Albers-Schönberg disease, in the malignant form, should not be confused with any other malady because of its characteristic symptoms. The benign type, in which many of the secondary manifestations are missing, is much more difficult to recognize. The outstanding diagnostic aid is a thorough radiographic examination of the entire skeleton. With this, a complete history and serologic and hematologic studies are extremely important.

Various causes can produce a generalized or a localized osteosclerosis which should not be confused with Albers-Schönberg disease. The following conditions should be considered:

1. *Syphilis*.—Some syphilitic lesions may cause thickening of the bone as well as the periosteum, but the base of the skull is rarely involved. Areas of destruction of bone may also be detected in the radiographs.

2. *Lead and Phosphorus Poisonings*.—Chronic lead and phosphorus poisonings cause more or less local sclerosing which improves when these agents are withheld. Necrosis of the jaw similar to that in osteopetrosis may result from phosphorus poisoning.

3. *Irradiated Ergosterol*.—Irradiated ergosterol and similar therapeutic agents in excessive amounts sometimes cause dense bands across the metaphyses of bones in children.

4. *Newborn Physiologic Osteosclerosis*.—In the newborn infant, physiologic osteosclerosis caused by the storage of calcium usually disappears within a few weeks.

5. *Fluorine Poisoning*.—Persons who have ingested large amounts of fluorine have been known to develop fluorosis, which presents a picture similar to Albers-Schönberg disease. Speder has observed patients in zones where the drinking water contains fluorides to have the following symptoms: malformation of the mandible, disturbance of dentition, osteomyelitis of the jaws, and anemia. The symptoms are common in osteopetrosis. When fluorine is consumed in abnormal quantities, it is accumulated and deposited in bones and teeth. A biopsy or necropsy would reveal calcium fluoride crystals in the Haversian system. This is caused by the fact that only a small amount of fluorine is eliminated, but just how this storage takes place is not fully understood. In fluorine intoxication the adjacent soft tissues are frequently calcified.

6. *Osteopoikilosis*.—Radiographs of osteopoikilosis reveal multiple spotted or mottled areas of sclerosis. These areas are scattered throughout the spongiosa of the epiphyses and

metaphyses of the long and flat bones. They are occasionally seen throughout the entire skeleton, but the skull, ribs, and vertebral column rarely display the condition.

7. *Eburnizing Osteitis*.—Eburnizing osteitis of Putti reveals an ivory-like density of a single bone usually localized in one extremity.

8. *Ivory Vertebrae*.—This is localized to sclerosis of one or two vertebrae.

9. *Condensing Osteitis*.—Condensing osteitis of Sicard shows bone changes associated with pain, usually limited to the lumbar region and pelvis. However, pathologic variations in the blood picture are lacking.

10. *Leucemia*.—In leucemia the alterations are infiltrative and destructive with more involvement of periosteum.

11. *Melorrheostosis*.—Melorrheostosis of Leri causes a distortion of the contour and a waxy appearance of the bones. It is usually limited to one or more bones of the extremities.

12. *Hodgkin's Disease*.—Bony changes of Hodgkin's disease may be similar to osteopetrosis, but a biopsy of a lymph node will readily distinguish between the two.

13. *Myelosclerosis*.—Myelosclerosis, also called myelofibrosis, osteosclerosis anemia, myelophthisic anemia, leucoerythroblastic anemia, and nonleukemic myelosis, displays diffuse, moderately increased density of the bone without transverse striations. Other symptoms consist of weakness, dyspnea, refractory anemia, splenomegalia, periostitis, and bone pains. Multiple fractures and optic atrophy are not present. It occurs in adults only. No familial or hereditary factor has been reported in the literature. The opacity of the bones in the radiographs is not comparable to Albers-Schönberg disease. The bone marrow is replaced by mild sclerosis or fibrosis.

14. *Osteogenesis Imperfecta*.—In osteogenesis imperfecta, also known as osteopsathyrosis, fragilitas ossium, periosteal aplasia, brittle-bones, and Lobstein's disease, the history of frequent fractures may be confusing. In this condition the density of the bones is decreased instead of increased. In fact, it seems to be diametrically the opposite of Albers-Schönberg disease.

15. *Leontiasis Ossea*.—This is a hyperplasia of the bones of the face and cranium leading to a lionlike facial appearance.

ETIOLOGY

Although Albers-Schönberg disease is of unknown origin, many etiological theories have been advanced. Among them are:

1. Defect in the germ plasm.
2. Mutation of the germ plasm "resulting in a dysplasia of that portion of the mesenchyme which, in its evolution, is destined to form the hematopoietic system."
3. Consanguinity has accentuated this Mendelian recessive.
4. Possibly a different group of etiological factors may exist in the benign cases of adults as compared with the malignant type found in children.

A condition similar to marble bones has been produced in experimenting on rats by prolonged injections of a concentrated extract of parathyroid principle. Similar results in animals have been produced experimentally with excessive amounts of ergosterol.

A few commentators have suggested that the ingestion of large amounts of fluorine may be the cause of Albers-Schönberg disease. If this could be substantiated, it should act as a warning to those who are advocating the use of fluorine in drinking water as a preventive measure for dental caries. A thorough investigation of patients in areas where fluorine is excessively ingested might reveal a striking correlation.

TREATMENT

Thus far, symptomatic treatment is the only means known to prolong the lives of these patients and relieve the secondary manifestations. The anemias may be improved by the injections of liver and iron and by transfusions as

indicated. Trauma should be avoided to prevent fractures and, likewise, every means possible should be instituted to ward off oral and general infections. Dental caries and periodontal disease ought to be cared for in their early stages.

The administration of ergosterol, cod-liver oil, calcium, and phosphorus is contraindicated as these might promote further growth of bone. Furthermore, all therapeutic measures to modify the mineral content of the bones, including the extracts of endocrine glands, have proved futile.

One author expresses the opinion that a partial parathyroidectomy might prove a valuable procedure.

Consanguineous marriages must be discouraged if either person is known to have the disease.

ORAL ASPECT

The oral aspect of this condition is important. The teeth usually erupt late and become carious early. The delayed eruption of the teeth is probably caused by sclerosing of the bones. However, the condition may also be due to some involvement of the tooth buds or to faulty general development. Dental caries is repeatedly mentioned in the literature as a cause of the premature loss of teeth. Other dental anomalies are noted, such as: hypoplastic teeth, partial anodontia, overgrowth of the mandible, prognathism, and progressive spreading of the teeth with resulting malocclusion.

Although periodontal infection in cases of Albers-Schönberg disease has rarely been reported, it has, no doubt, been overlooked or not recognized as there are many factors which point to its existence even in an advanced form. Osteomyelitis of the maxilla and mandible is one of the most dreaded and dangerous hazards to these patients. It has resulted in death in five instances following tooth infection and in complete sequestration of the mandible in two cases.

Dental caries and necrosis of the jaws in these cases have been attributed to constriction of the bony canals of the alveolar arteries. As a result, the decrease in the blood supply favors the spread of infection throughout the jaw-bones.

CASE 1

G. B., single white man, 19 years of age, was referred to my office on April 1, 1944, for dental examination. He appeared to be in good health and was well proportioned except for a large mandible.

Family History.—The patient had one sister, 21 years old, living and well. She had no bony abnormalities which could be recognized by complete clinical or radiographic examinations. The patient's only brother, 23 years old, was serving in the Armed Forces. The mother stated, "This brother cannot swim or float," apparently because of the specific gravity of his bones. The patient's father, a toolmaker, died of tuberculosis at the age of 36 years. Although he was ill only ten days, the disease was known to have existed for ten years. The mother is the patient described in Case 2. No history of consanguinity in the family was revealed.

Past History.—The patient was born spontaneously at full term. He was breast fed for nine months and has always enjoyed a well-balanced diet. An umbilical hernia was detected shortly after birth, but this disappeared without treatment at the age of 4 years. Between his fifth and seventh years, he had measles, mumps, and chicken pox. His tonsils and adenoids were removed at 7 years of age. When he was 13 years old, he had a severe infection of the upper part of the respiratory tract. Recently he started smoking moderately, but he does not use alcoholic beverages. He has participated strenuously in many sports, including football, and has been in two serious automobile accidents without ever fracturing a bone. No mental retardation was observed; he graduated from high school at the age of 18 years.



Fig. 1.—Case 1. Upper teeth.

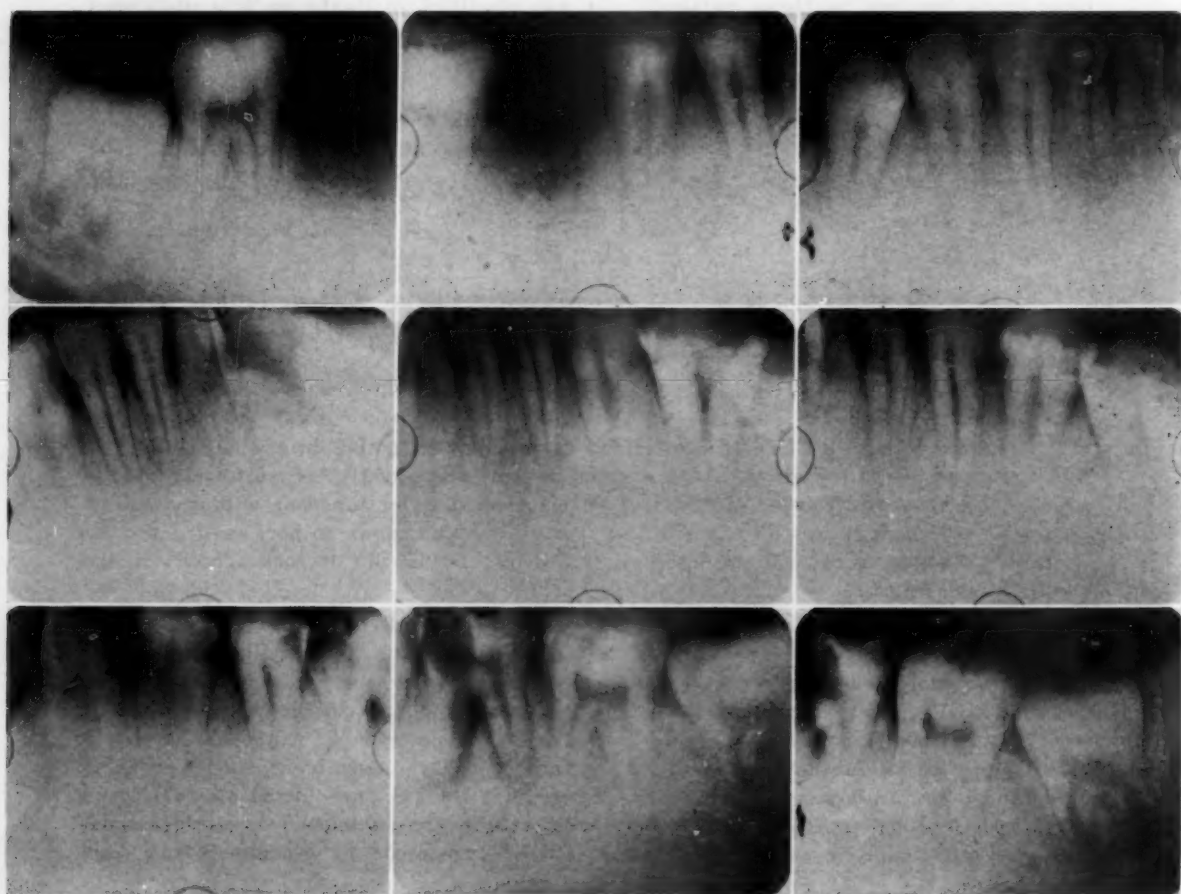


Fig. 2.—Case 1. Lower teeth.

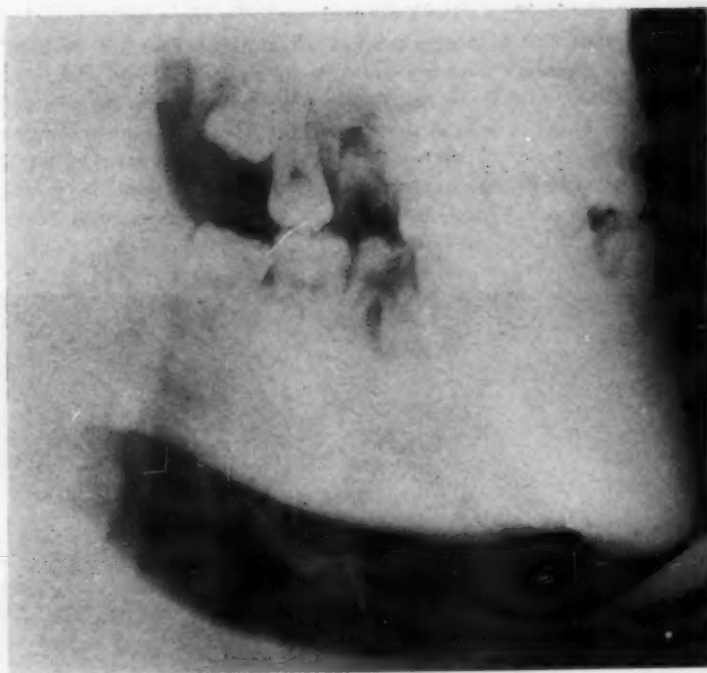


Fig. 3.—Case 1. Lateral left jaw.



Fig. 4.—Case 1. Lateral skull.

Dental History.—No deciduous teeth erupted until the boy was 15 months old, the others following normally. There was nothing abnormal about the exfoliation of his deciduous teeth nor the eruption of his permanent ones. He has never had a toothache.

Physical Examination.—On July 19, 1943, a physical examination revealed the patient's height to be 71 inches, and his weight, 161 pounds. No significant findings were disclosed other than a hydrocele. Grossly, his eyes were normal, his vision being 20/15 in each eye. Ophthalmoscopically, the media were clear and the fundi normal in appearance. There was no pallor of the optic nerve head in either eye. Neither nystagmus nor strabismus was present.



Fig. 5.—Case 1. Pelvis.

Radiographic Examination.—The inner and outer tables of the skull were thickened and lacking in structural details; the diploe was not visible. The density of the frontal, parietal, occipital, and other basilar bones, including the petrous apices, was greatly increased. The clinoid processes were considerably thickened but the sellar cavity was normal in size. In the facial area, the only nasal accessory sinuses visible were the ethmoids and small antrums. In comparison, the mandible was only moderately thickened, the trabeculae being visible. The vertebrae and pelvic girdle were thickened throughout, the pelvic apophyses showing normal development for his age. In the long bones and hands the cortex was thickened with the medullary portions narrowed showing a hazy delineation. No transverse or longitudinal stratification was noted. The fingers showed a tendency to lengthening and shapelessness with the shafts approaching the diameter of the heads and bases. In the

phalanges the cortex and medulla were not sharply defined. The increased density was fairly evenly distributed except that the bases were denser. The terminal tufts were small. No fractures or calcified vessels were detected. A complete radiodontic examination disclosed considerable sclerosis of the jaws. All radiographs were made at kilovoltages considerably above those normally employed for the part.

Laboratory Report.—The electrocardiographic report was within normal limits. The basal metabolic rate was +9. The sedimentation rate was 4 mm., Cutler method. Blood Wassermann was negative; blood sugar fasting, 105 mg. per 100 c.c. of blood; blood urea nitrogen, 19 mg.; serum nonprotein nitrogen, 37.5 mg.; serum protein, 5.8 Gm.; serum albumin, 3.2 Gm.; serum globulin, 2.6 Gm.; blood chlorides, 406 mg.; blood calcium, 9 mg.; blood cholesterol (plasma), 222 mg.; blood grouping, international type O; icterus index, 4; erythrocyte count, 4,770,000; leucocyte count, 8,600; hemoglobin, 81 per cent or 12.5 Gm. per 100 c.c. of blood, Haden Hauser method; color index, 0.8; polymorphonuclear leucocytes, 65 per cent; lymphocytes, 35 per cent; eosinophiles, 2 per cent; neutrophiles: band cells 2, segmenter cells 61; coagulation time, 2 minutes; bleeding time, 1 minute; acromia, slight. Urinalysis was as follows: light amber color, slightly cloudy, aromatic odor, acid reaction; specific gravity 1.022; albumin, sugar, and indican, none found; white blood cells, few; epithelial cells, round and squamous; mucus present; no casts. The calcium precipitability in the urine was within normal limits. The hydrogen-ion concentration of the saliva taken by the glass electrode meter was 7.15.

Dental Examination.—The orifice of the patient's mouth was small and his lips were dry. Both maxillary permanent lateral incisors were congenitally missing; the deciduous lateral incisor was still present on the left side. Hypoplastic pits were visible in the anterior teeth. The mouth was fairly clean and only a small amount of calculus was detected. The mandibular right first molar had been extracted because of caries and the other three first molars were badly broken down with a small fistula present on the mandibular left side. Many carious areas were apparent in the other teeth. Nevertheless, his teeth were better than average considering that the patient had had no conservative dental work done. The mucous membranes were normal. There was a tendency toward a groove in the palate with one side slightly more prominent; however, no torus existed. The tongue was normal in size, color, and texture. The mandible was excessively developed, especially vertically, and a Class III (Angle) malocclusion was observed.

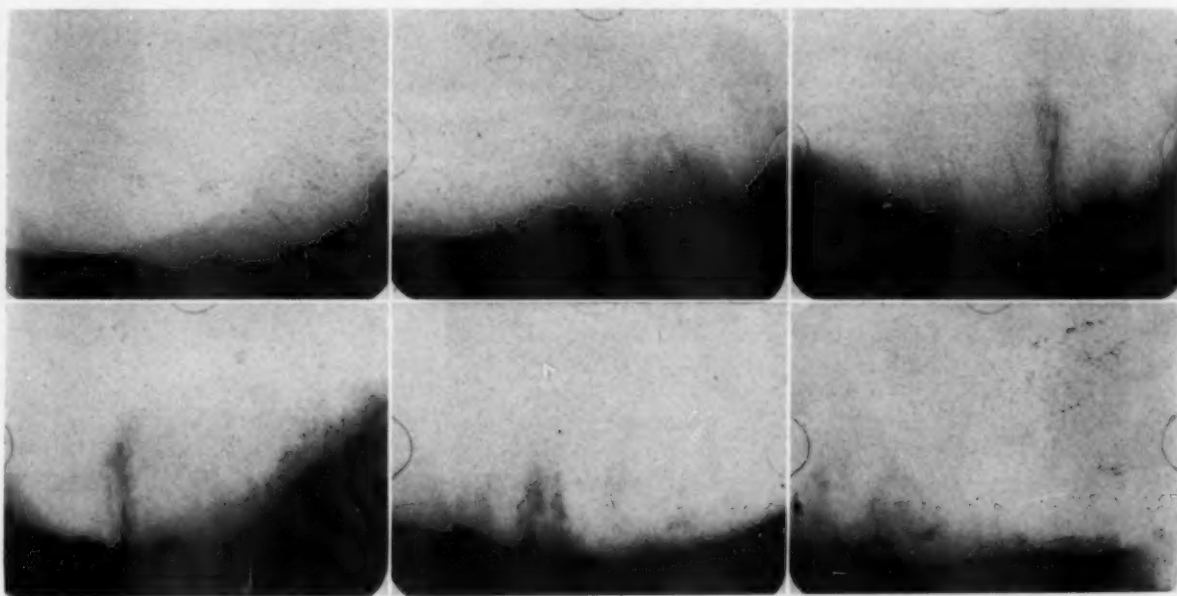


Fig. 6.—Case 2. Upper jaw.

CASE 2

M. B., mother of man in Case 1, presented herself for dental examination on April 1, 1944. She was a widow, 55 years of age.

Family History.—She had no brothers or sisters. Her mother died at the age of 58 of Bright's disease. Her father, who was a machinist, is believed to be dead (no information

obtained). Her maternal grandmother died at the age of 84 of "old age," and her maternal grandfather died at the age of 52 of pneumonia. Her paternal grandmother died at the age of 82 of a "heart attack," and her paternal grandfather died about the age of 70, cause unknown. Her husband was the father of the boy in Case 1. The patient stated that she had always lived in upper New York State, and, as far as she knew, her parents had also resided in New York State where no excessive amounts of fluorine have ever been found, either in the soil or in the drinking water.

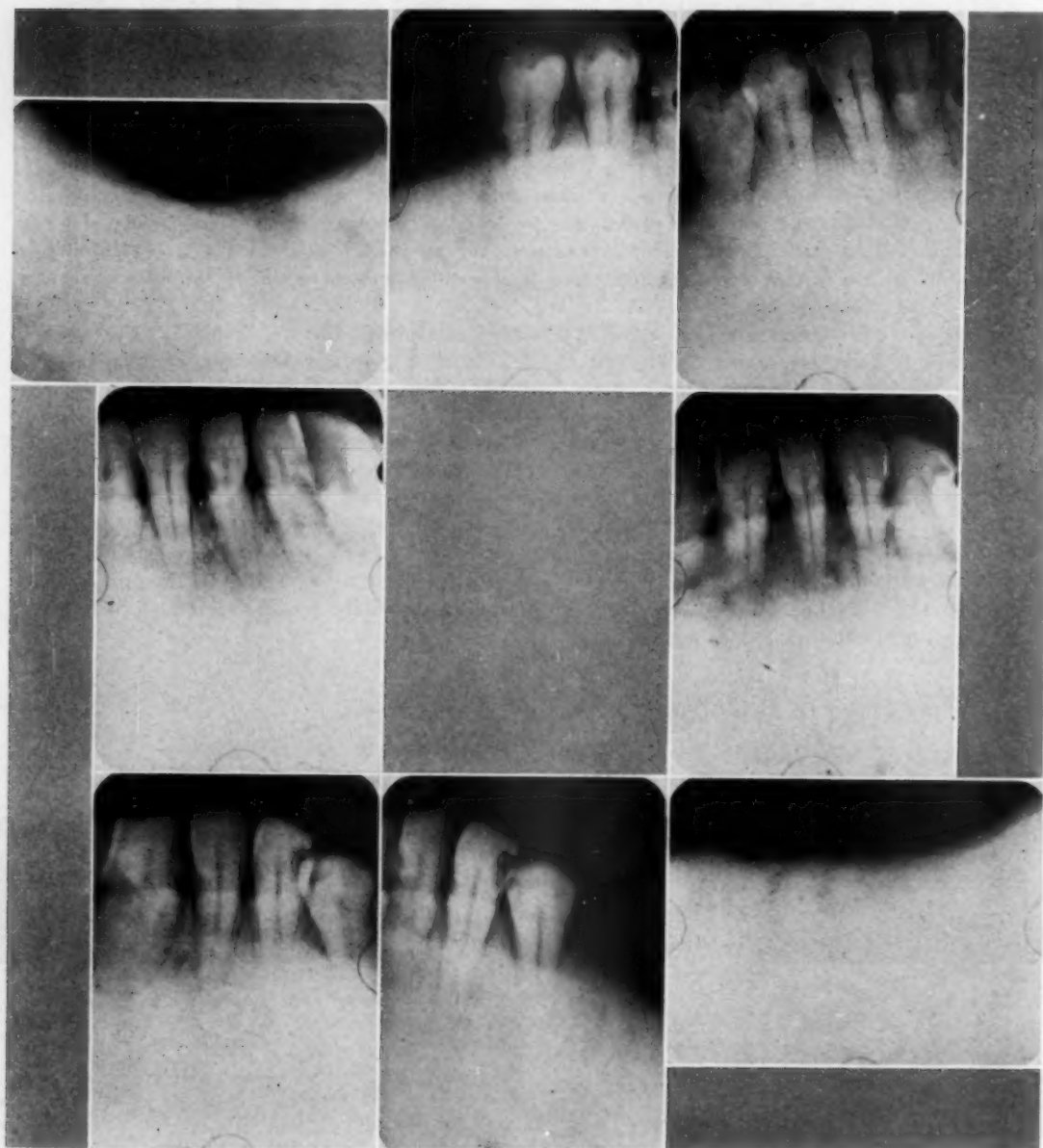


Fig. 7.—Case 2. Lower jaw.

Past History.—The patient, a practical nurse, has enjoyed unusually good health throughout her life. She has never fractured a bone, had an operation, nor been ill, except for chicken pox and measles when she was 12 years old, and an iritis when she was 31 years old. This was a severe infection; the left eye did not heal for six months and, in the right eye, the sight was lost completely. At the present time this functionless eye protrudes. Her menstrual periods and menopause were normal. She had three pregnancies complicated by severe vomiting, but all three deliveries were normal, and each child was breast fed for nine months.

Six months ago she went to a physician complaining of loss of weight. A radiograph of the intestinal tract was among the diagnostic procedures instituted. This, of course, brought to the attention of the radiologist and internist the uniform white appearance of the bones.

Dental History.—The dental history was more or less irrelevant until five years ago when all of her maxillary teeth and the mandibular posterior teeth were removed. The extractions were difficult and the healing slow. Four months after extraction of teeth, the maxillary ridge was so irregular and presented so many protuberances that the patient was sent by her dentist to an oral surgeon for a partial alveolectomy. When the operator retracted the soft tissues and started to excise the bony tissue, he met with much difficulty. Before the operation was completed, two rongeur forceps were broken.

Physical Examination.—A physical examination on July 1, 1943, revealed the patient's height to be 6 feet, and her weight, 152 pounds. Her facial appearance was pale. The blood pressure was 124/80. The right eyeball bulged; the pupil was small, and irregular scars were present on the cornea. Keratitis was noted on the right temporal side. The pupil in the left eye reacted normally. A sense of fullness was detected in the hepatic flexure region but there was no definite mass. External hemorrhoids were visible. Further examination clinically showed no pathologic changes.

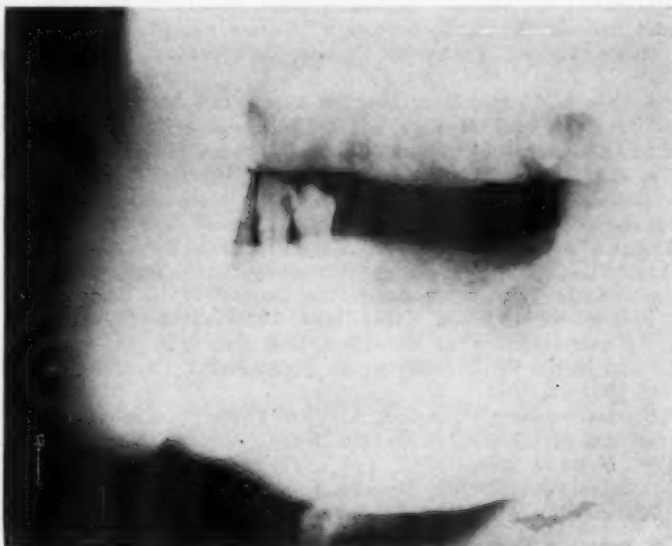


Fig. 8.—Case 2. Lateral left jaw.

Radiographic Examination.—The skull was generally the same as that of the patient in Case 1 except that the greatest density was in the occipital and frontal areas, the latter showing hyperostosis frontalis. The posterior clinoid processes were not as enlarged as in her son but the density of the mandible was greater. The vertebrae and pelvic girdle showed increased density although not as pronounced as in her son, while the density in the long bones and hands was practically the same. There were no fractures or calcified vessels detected. A complete intraoral radiodontic examination revealed unusual density of the maxilla and mandible. Areas of condensed bone resembling dental roots were visible in the lateral radiographs of the jaws. All radiographs were made at kilovoltages considerably above those normally employed for the part.

Laboratory Report.—The sedimentation rate was 7 mm. (first hour), 11 mm. (second hour); erythrocyte count, 4,340,000; leucocyte count, 7,700; hemoglobin, 85 per cent; color index, 0.98; polymorphonuclear leucocytes, 50 per cent; small lymphocytes, 46 per cent; eosinophiles, 1 per cent; transitionals, 3 per cent; blood Wassermann, negative. Examination of stool revealed slight amount of occult blood. Urinalysis was as follows: acid reaction; specific gravity, 1.020; albumin, negative; sugar, negative; pus, 3-5.

Dental Examination.—The upper jaw was edentulous, a full acrylic denture being worn fairly successfully. The seven anterior teeth which were present on the lower jaw revealed four carious areas. Considerable amounts of calculus were observed, and, in turn, a well-established case of periodontoclasia. No other abnormal conditions in her mouth were noted.

DENTAL COMMENTS

Two cases of the benign type of Albers-Schönberg disease are reported. Both are without the majority of the secondary manifestations. Because of the seriousness of dental infection and the difficulties encountered in surgical procedures of the jaws, more cases of this and other bone diseases should be brought to the attention of dentists. Too few case reports have been presented in the dental literature. The dental profession should be interested in osteopetrosis because the ingestion of too much fluorine has been suggested as a causative factor.

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TERATOID PARASITES OF THE MOUTH
(EPISPHEOIDS, EPIPALATI [EPURANI], EPIGNATHI)

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THE teratoid parasites of the mouth have long attracted the attention of the teratologists. They have been thoroughly analyzed by Ahlfeld (1875), Arnold (1888), Marchand (1897), and Schwalbe (1907). They have been used by Schwalbe as a basis for the development of his concept of the "teratogenetic termination period."†

At first these parasites were believed to originate in the upper jaw and therefore were called *epignathi* (upon the jaw) (I.G. Saint-Hilaire).‡ Later it was pointed out that most arose at the hard palate and some were attached to the roof of the pharynx (Ahlfeld, Arnold). When reviewing 22 cases reported in the literature since 1922 available here, it was found that 11 were attached to the sphenoid bone, 6 to the lateral wall of the epipharynx (around the mouth of the Eustachian tube or the area of Rosenmüller's groove), 2 to the soft palate, and 1 to the hard palate. Since the recent descriptions seem to be more accurate than some of the older ones, it appears that only few oral parasites arise primarily in the jaw. Hence, they should not be called *epignathi* (except in the few cases where they arise in the jaw); but if it is desired to retain the nomenclature, the parasites of the palate should be called *epipalati* or *epurani* (upon the palate), and those of the sphenoid *episphenoids* (upon the sphenoid).

It was proposed by Marchand to speak of *uranopagus* and *sphenopagus* (fixation to the palate and sphenoid). These would be excellent terms if *pagus* were not generally used to designate *symmetrical* double monsters only. If all oral parasites were *asymmetrical* double monsters, as this writer believes, we could speak of *sphenopagus parasiticus*, *palato-* or *uranopagus parasiticus*, and *gnathopagus parasiticus*—for these are recognized terms for *asymmetrical* double monsters.

While episphenoids develop mostly into the pharynx and mouth, in some cases they grow into the cranium and thus become *enceranii*. In fact, a considerable number of *enceranii* are episphenoids that have grown into the cranium instead of into the pharynx and mouth. In rare cases episphenoids develop in both directions; since these are of considerable teratological importance, it seems to be indicated to report an unusual case which was recently observed in the Philadelphia General Hospital.

CASE REPORT

Philadelphia General Hospital Unit History No. 150794, Autopsy No. 48197.

Clinical.—The stillborn infant, a Negro female, was delivered by cesarean section on Nov. 23, 1944. The mother, an 18-year-old Negro primipara, had menstruated regularly since she was 13 years old. The last menstruation occurred five months prior to delivery on June 22, 1944. On admission to the hospital, the patient showed a large distended uterus suggesting polyhydramnios. There were no fetal heart beats. The polyhydramnios was confirmed

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†The latest possible moment when a given malformation may have developed.

‡Quoted by Ahlfeld (1875).

during operation. Since bleeding could not be stopped, supravaginal hysterectomy was performed. Recovery was uneventful.

Repeated Kline tests were negative.

Post Mortem.—Gross examination of the fetus revealed a Negro female weighing 3 pounds, 14 ounces, with a crown-to-heel measurement of 38 cm. The head was very large; it measured 10 cm. in lateral diameter and 12 cm. in anteroposterior diameter. From the mouth protruded a mass measuring 5 cm. in height, 8 cm. in width and 4 cm. in depth. The mass was irregular in shape; it was covered with what appeared to be skin; there were no distinct limbs present.

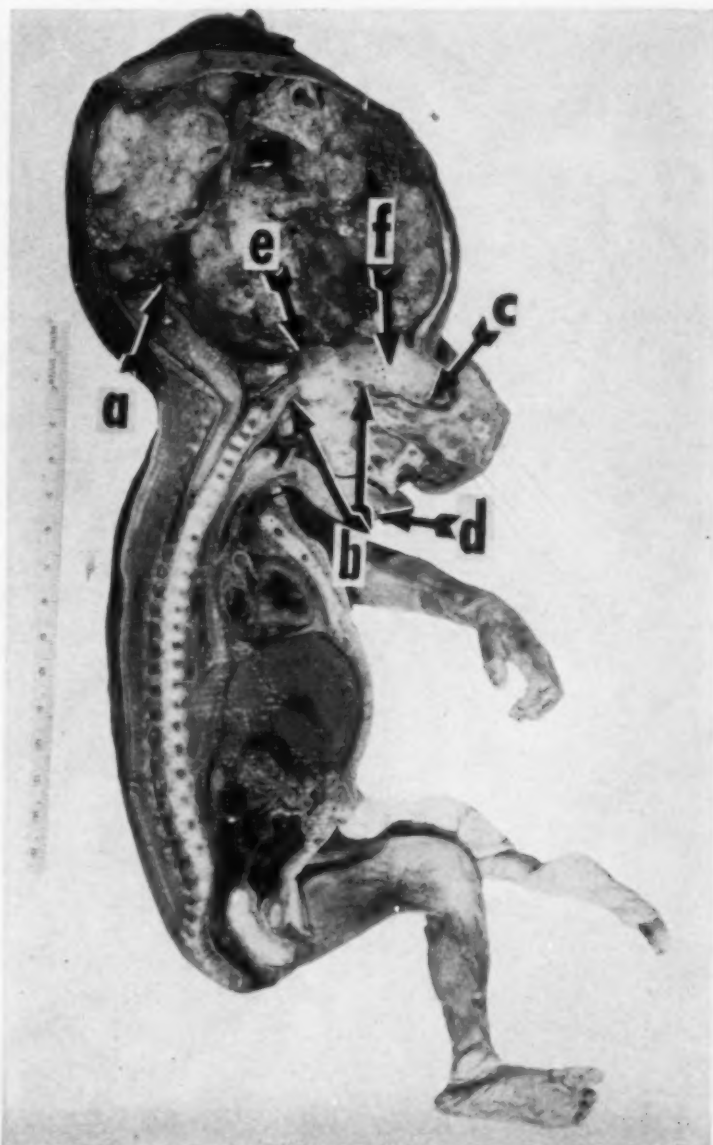


Fig. 1.—The left half of the fetus showing the position of the parasite. The brain of the autosite is greatly compressed (a). Upper jaw (c), lower jaw (d), dorsum sellae (e), and lamina perpendicularis (f) are well preserved. The area of attachment of the oral portion of the parasite to the sphenoid bone of the autosite is indicated by a pair of arrows (b).

After fixation, the fetus was frozen and cut in two.* Inspection of the left half of the fetus (Fig. 1) reveals a large lobulated mass filling most of the cranium. The brain is greatly reduced in size; it lies in an area immediately over the foramen magnum (a). The mass filling the pharynx and mouth (as well as the nose) is attached to the region of the sphenoid bone (b). Both the upper jaw (c) and the lower jaw (d) are well preserved. The base of the skull seems to be largely intact: the clivus and its dorsum sellae (e) are well developed;

*We are indebted to Dr. O. V. Batson for cutting the fetus with his rotating saw. The excellence of his work is documented in Fig. 1.

the lamina perpendicularis (*f*) is complete; there is no sphenoid sinus; this is formed only some time after birth.

Microscopically, it was found that the upper and anterior surfaces of the intraoral portion of the parasite were covered with well-differentiated skin consisting of keratinized epidermis, hair, sebaceous glands, and sweat glands (Fig. 2, *A*). The lower surface showed nonkeratinized epidermis such as one observes in the vagina; there were no hair follicles, sebaceous glands, or mucous glands (Fig. 2, *B*). The posterior surface near the roof of the mouth revealed a single layer of well-differentiated ciliated epithelium, probably nasal epithelium of the autosite* (Fig. 2, *C*).

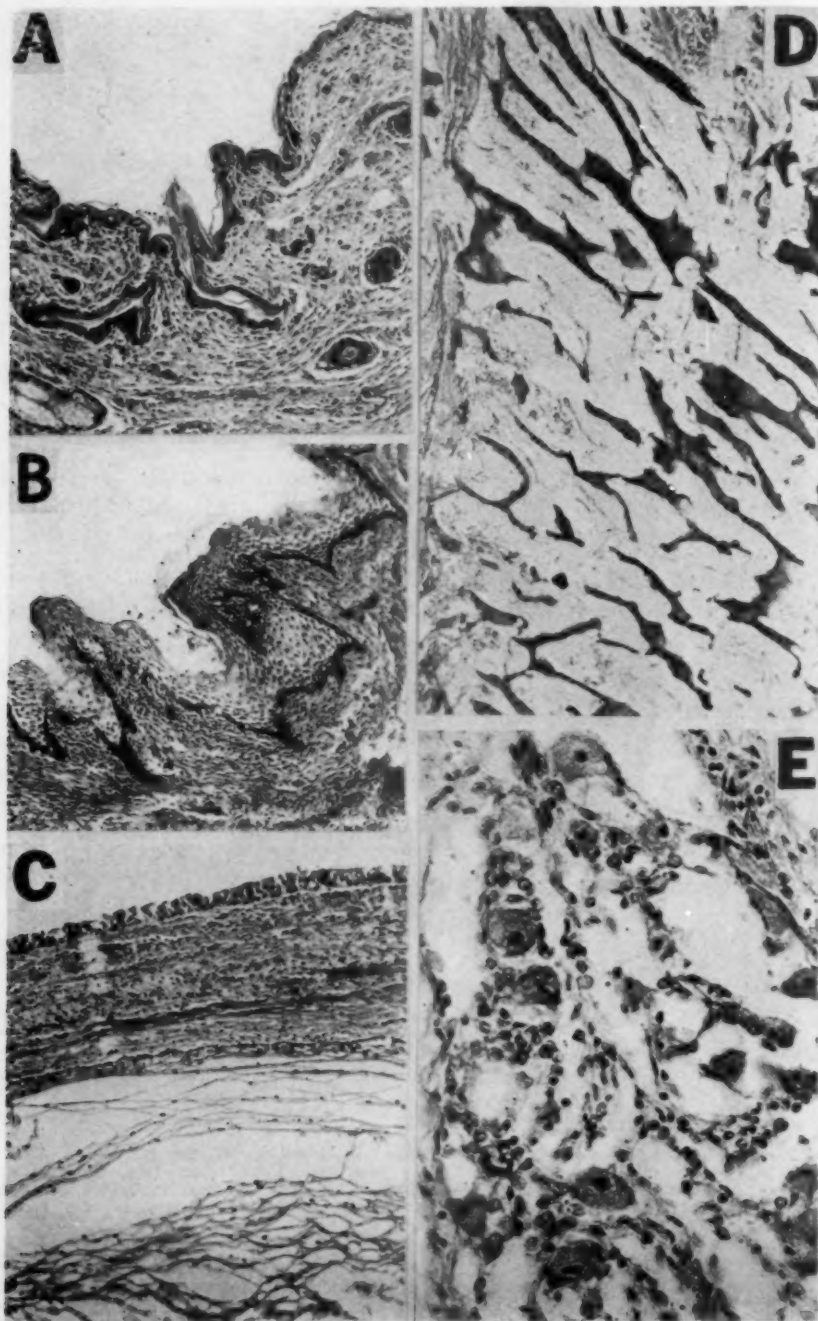


Fig. 2.—Sections from the oral and pharyngeal portion of the parasite showing (A) skin with epidermis, hair follicles, and sebaceous glands ($\times 90$), (B) nonkeratinized epidermis ($\times 90$), (C) ciliated epithelium ($\times 90$), (D) bone tissue ($\times 55$), and (E) ganglial cells ($\times 400$).

*An autosite is the better developed twin of an asymmetrical double monster; it is the one upon which the parasite lives.

The interior of the intraoral portion of the parasite consisted mostly of connective tissue, cartilage, and both membranes and cartilagenous bone (Fig. 2, *D*). It also showed some fat tissue, some nerves, occasional cysts lined with squamous epithelium, and a few ganglia with well-differentiated ganglial cells (Fig. 2, *E*). Near the roof of the mouth and in the intranasal portion of the parasite we found brain tissue, including spaces lined with branching blood vessels covered with a single layer of cuboidal epithelium resembling choroid plexus (Fig. 3, *A*).

The portion of the parasite that filled the cranium was partially covered with skin including hair follicles. Other parts were covered with oral mucosa showing nonkeratinized epidermis as well as mucoid glands (Fig. 3, *B*).

The interior of the intracranial portion consisted of a rather bewildering mixture of poorly organized tissues: there was a good deal of brain tissue; there was a large primordium

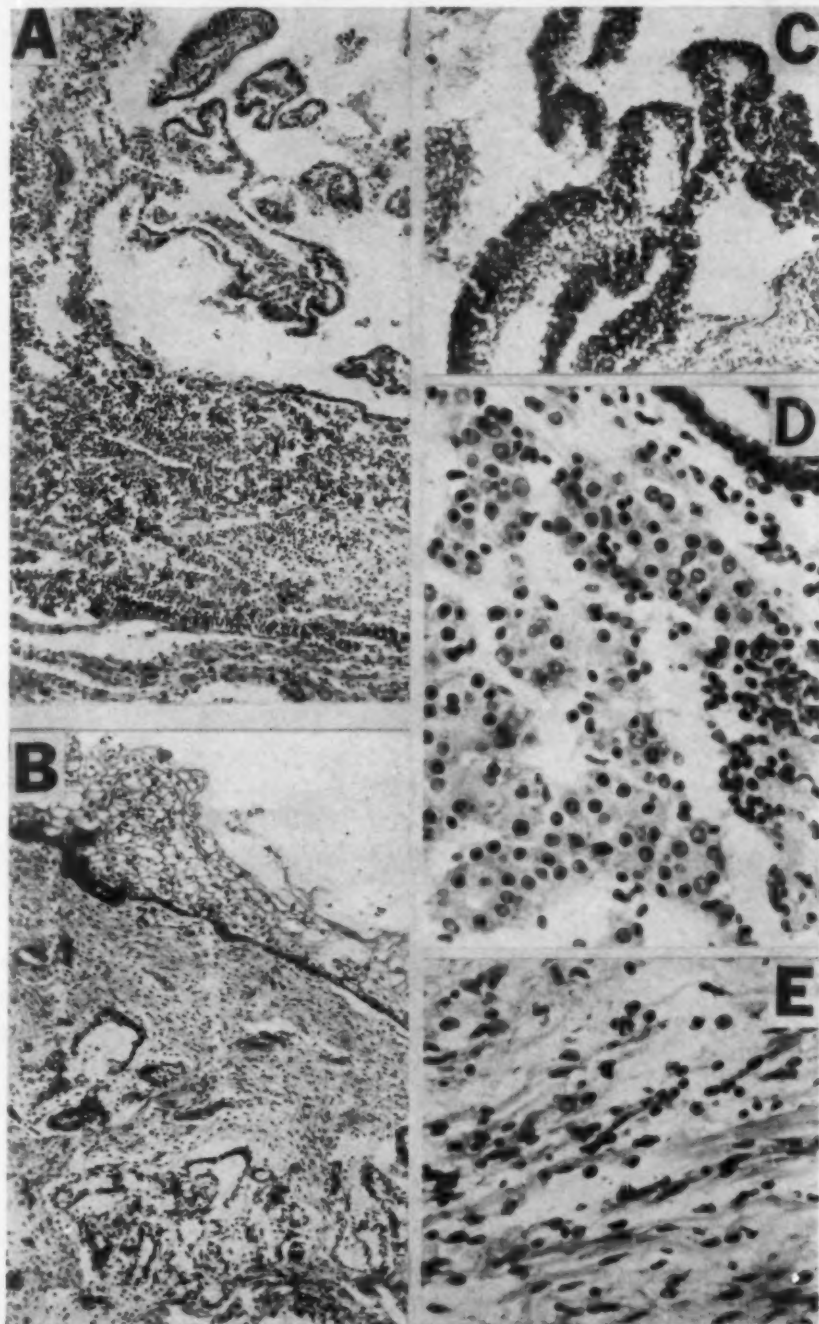


Fig. 3.—*A*, Section from the intranasal portion of the parasite showing brain tissue and choroid plexus ($\times 90$). *B* to *E*, Sections from the intracranial portion of the parasite showing (*B*) oral mucosa with mucous glands ($\times 100$), (*C*) retinal primordium ($\times 100$), (*D*) liver tissue ($\times 400$), and (*E*) musculature ($\times 100$).

of an eye including retinal pigment (Fig. 3, *C*); there was abundant liver tissue including bile ducts (Fig. 3, *D*); there was muscle tissue (Fig. 3, *E*). We also observed glandular spaces lined with various epithelial elements as well as a great deal of poorly differentiated embryonal material the nature of which was not identified. Parts of these tissues were necrotic and infiltrated with lime salts.

The base of the skull of the autosite first presented some difficulty as it was not possible to find a canal connecting the intracranial and intraoral portions of the parasite; the two parts were separated by the sphenoid bone as is well shown in Fig. 4, *A*, *a*. But semiserial sections then revealed that there was unorganized brain tissue above and below this bone (Fig. 4, *A*, *b*, and *c*), and there was brain tissue, instead of bone marrow, in the interior

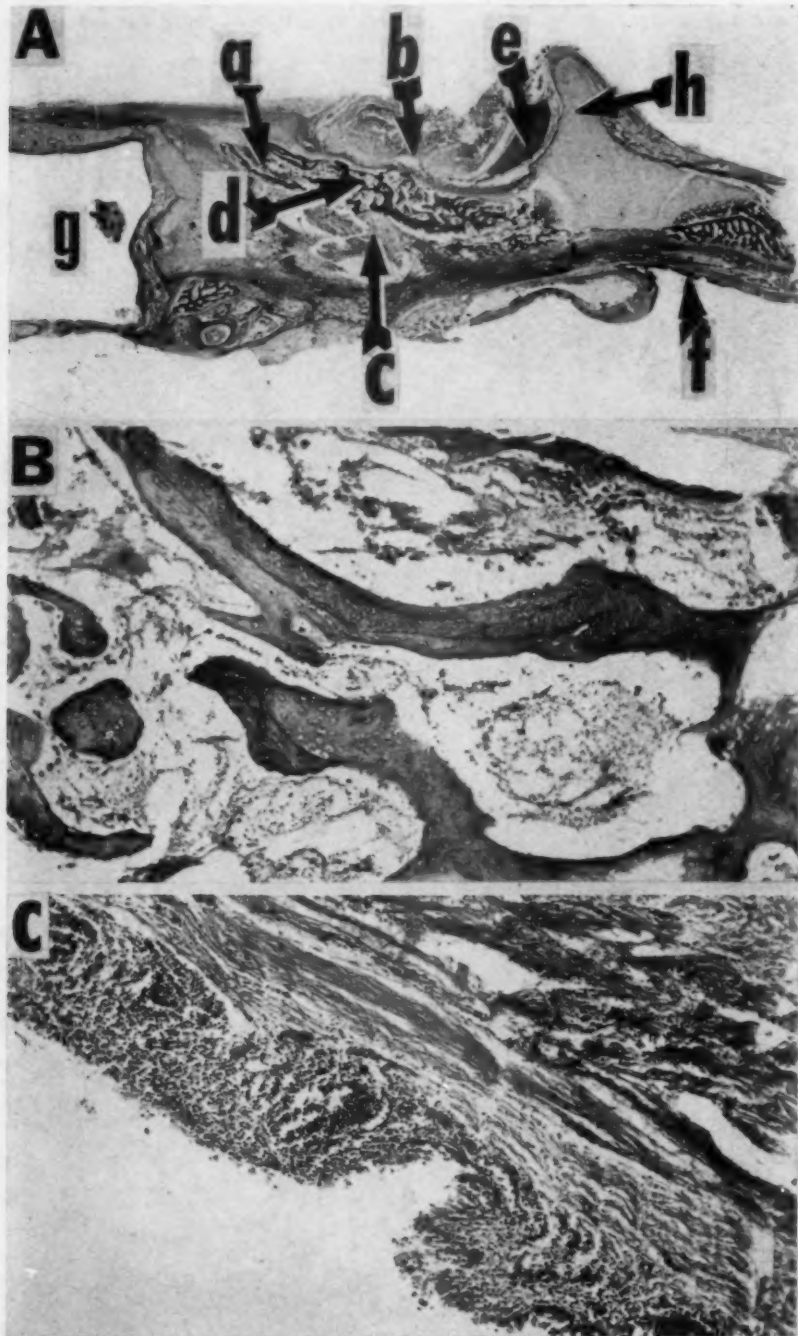


Fig. 4.—*A*, Section through the base of the skull of the autosite ($\times 8$) showing (*a*) the sphenoid bone, unorganized brain tissue (*b*) above, (*c*) below, and (*d*) within the sphenoid bone, (*e*) the incompletely developed anterior lobe of the pituitary gland, (*f*) the pharyngeal tonsil, (*g*) the nasal cavity, and (*h*) the clivus with the sella turcica. *B* is a higher magnification of the sphenoid bone showing brain tissue instead of hematopoietic marrow ($\times 55$); *C* is a higher magnification of the pharyngeal tonsil ($\times 90$).

of the sphenoid bone (Fig. 4, A, d and Fig. 4, B). The place where this occurred was identified as the base and front of the sella turcica, i.e., the area of the craniopharyngeal canal. The anterior lobe of the pituitary gland was found to be incompletely developed and pressed against the back of the sella turcica (Fig. 4, A, e); the posterior lobe was not discovered. The attachment of the intraoral portion of the parasite was found to extend from the pharyngeal tonsil (Fig. 4, A, f, and Fig. 4, C) to the nose of the autosite (Fig. 4, A, g). The clivus was not malformed (Fig. 4, A, h).

COMMENT

The history of the teratoid parasites of the pharynx and mouth has been well described by Ahlfeld (1875). In 1880 he found forty cases in the literature; some were attached to the autosite by an umbilical cord. Since the higher-developed parasites showed brain tissue in the upper pole and extremities in the lower, Ahlfeld concluded that they were double monsters. Since they had no heart, he classified them with the acardiaci. As to the pathogenesis of these malformations, Ahlfeld postulated that of two head-on developing twins one was caught under the anterior cerebral vesicle of the other while it was bent to form the pharyngeal space; the parasite thus became secondarily attached to the autosite at the junction of notochord and pharyngeal membrane where sphenoid bone and pituitary gland later made their appearance.

The publications of Ahlfeld were soon followed by a notable contribution of Arnold (1888) who distinguished four groups of oral parasites. A first group (of 7 specimens) consisted of usually pedunculated, pear-shaped dermoids attached to the palate or epipharynx, consisting chiefly of fat tissue covered with skin including hair, sebaceous glands, and sweat glands; these were called *hairy polyps*. A second group (of 7 specimens) comprised larger parasites which mostly protruded from the mouth and contained derivatives of all three germinal layers. A third group, his fourth group* (of 20 cases), consisted of incomplete double monsters showing grossly recognizable extremities, fingers, eyes, or an umbilical cord. As to the pathogenesis of these malformations, Arnold came to the conclusion that the members of his first group were no true double monsters, but tissue malformations due to dislocation of parts of the ectodermal primordium of the pituitary gland; and his second group could also be explained by dislocation; but his fourth group consisted of true double monsters; they were "genuine epignathi"; in fact, he spoke of *foetus in foetu*. Arnold thus distinguished between "autochthonous and heterochthonous teratomas," the autochthonous being of monogerminal, the heterochthonous of bigerminal origin.

A third comprehensive study to be mentioned here is the one by Schwalbe (1907) who, like Arnold, divided his "epignathi" into four groups. His first group comprised the very rare twinlike monsters connected with the autosite by an umbilical cord. His second group consisted of less well-organized monsters showing recognizable extremities or sexual organs; these monsters were rare too. His third group comprised the unorganized teratomas containing derivatives of all three germinal layers. His fourth group included the dermoids or hairy polyps of Arnold; these were the most common "epignathi." The first and second groups of Schwalbe thus corresponded roughly to the fourth group of Arnold; while the fourth group of Schwalbe was the same as the first group of Arnold.

As to the pathogenesis of the various groups, Schwalbe, like Ahlfeld, believed that all arose by the same mechanism. But while Ahlfeld had thought that they were due to twin formation, Schwalbe felt that they were caused by

*Arnold's third group (of 4 specimens) is a miscellaneous group consisting of teratomas which have in common only that they contain well-developed teeth.

dislocation of germ material; they differed only in the time when the dislocation occurred: the teratogenetic termination period of the highly developed parasites was early during development, may be as early as the stage of gastrulation, while that of the hairy polyps was the third week when the primitive pharyngeal membrane made its disappearance.

During the last forty years, distinguished studies of the oral parasites have not been forthcoming. Reviews of the literature were furnished by Feller (1928) and Gerlinghoff (1928); a review of 62 hairy polyps was presented by Oppikofer (1932); the French and Italian literature was well reviewed by di Vestea (1937).

Higher-developed pharyngeal and oral parasites have recently been described by Gerlinghoff (1928); Strachau (1930), Wen (1933), and Bottiroli (1942). Hairy polyps (not included in Oppikofer's publication) have recently been reported by Nather (1922), Lichtenstein (1922), Bulson (1926), Schwarz (1928), Feller (1928), Santi (1929), and Friedman (1931), as well as by Hankins and Harding (1932), Stokoe (1937), di Vestea (1937), and Howarth (1938). These bring the number of known cases of hairy polyps up to 73.*

It is noteworthy that most of the higher-developed oral parasites were found in stillborn fetuses or newborn infants, while most hairy polyps were observed in living children or adults. Of 22 higher developed parasites collected by Arnold, 14 occurred in fetuses, 5 in newborn infants, and 3 in infants that died one to five days after delivery; of 7 hairy polyps, one was found in a newborn, 3 in infants from 1 to 6 months, 2 in children of 3 and 13 years, and one in an adult of 20 years. Of 3 highly developed parasites described during the last twenty years, all occurred in fetuses; of 14 hairy polyps observed during this period, one was seen in a fetus, one in a newborn infant, 3 in small infants 3 to 11 weeks old, 5 in older infants 3 to 14 months old, 3 in young people 14 to 23 years old, and one in an old man of 66 years.

It is also noteworthy that there was a marked preponderance of the female sex.

If we turn now to the encephali, we find a much briefer history. Ernst (1909) in a comprehensive review of the malformations of the nervous system reported only 10 dermoids and teratomas in the brain. Hosoi (1930) recorded 41 encephali: 19 were located in the epiphysis, 9 in the hypophysis, and the remaining in the brain, the cerebellum, and the ventricles. Manca (1938) collected 53 encephali: 19 were situated in the epiphysis, 12 in the hypophysis, 9 in the diencephalon, 4 in the metencephalon, and 3 in the telencephalon. All encephali arose at or near the midline.

Of the parasites showing both oral-pharyngeal and intracranial portions, finally, we found only six cases in the literature:

1. Wegelin (1861)† described a large intraoral parasite which, through a defect in the middle and anterior portion of the sphenoid bone and in a part of the lamina cribrosa, was connected with four cysts which were located within the cranial cavity. There was absence of both the soft and hard palate.

2. Breslau and Rindfleisch (1864) reported a large intraoral mass which through the basis cranii in the area of the sella turcica, by a round stalk, was connected with an intracranial mass which contained two eyes and at least seven extremities. The pituitary gland was completely absent.

*For the sake of completeness, it may be mentioned here that the present series of the *Index Medicus* (since 1927) contains references to 6 additional cases, namely those reported by Nikolaeff (1928) (epignathus), de San Pio (1929) (pharyngeal teratoma), Malan (1932) (tumor intramural palatini), d'Avino (1940) (hairy polyps), Droop (1940) (hairy polyp), and Ito (1941) (teratoma of nasopharynx). Since these were not available to the writer, they could not be included in this study.

†Quoted by Ahlfeld (1875).

3. Arnold (1870) observed a 6-day-old child with a large intraoral parasite which through "the area of the right foramen ovale" was connected with a walnut-sized intracranial mass which "reached" the back of the sella turcica and the clivus. The hard palate was split, while the soft palate was part of the intraoral portion of the parasite. The sella proper or the pituitary gland were not described. A drawing attached to the report makes it appear possible that the connecting stalk of the two portions passed through the area of the cranio-pharyngeal duct.

4. Mueller (1881) studied a 7-month-old female fetus showing a large intraoral teratoma which was connected with the left palate and cheek, the tongue, and the epipharynx. The left portion of the nose was also involved. There was an encranium of hazelnut size attached to the right middle cranial fossa. The pituitary gland was well formed. Though no connection was found between the intraoral teratoma and the encranium, Mueller felt that they were parts of one parasite; the connecting stalk had early during development been destroyed by the vigorously growing basis cranii. That this was the course of events was borne out by the observation that the encranium and the uppermost portion of the oral teratoma were histologically identical.

5. Schuekry (1923) gave a very brief description of a female newborn infant showing an encranial teratoma of the size of a child's fist attached to the right side of the sella turcica. Through the basis cranii it was connected by a dense fibrous tissue with a cherry-sized tumor in the epipharynx.

6. Kraus (1929) observed a female fetus with a large intraoral teratoma that through the patent craniopharyngeal canal, by a 7- to 8-mm.-long stalk, was connected with a small intracranial mass which was located in the area of the pituitary gland. The adrenals were found to be normal.

It is noteworthy that in all cases where the sex was registered, including our own case, the autosome was a female. It seems that with the exception of Arnold's case, all were stillborn.

From the descriptions given above as well as from our own case, it appears that the intracranial and intraoral portions of our combined oral-pharyngeal-intracranial parasites were all connected through the sphenoid bone, or more precisely, through the craniopharyngeal canal. This seemed to be true also in Arnold's case, the description of which is a little vague. If we consider this situation as well as the fact that a good many of the encranii reported in the literature were found to spring from the pituitary gland, and most of the pharyngeal and oral parasites were attached at or near the sphenoid bone; if we further consider that all encranii, and pharyngeal and oral parasites were found to arise at or near the midline, and all transitions were found between the hairy polyps and the twinlike double monsters, as all transitions were observed between the oral parasites and the encranii; if we consider all these facts, it suggests itself that all these malformations have a common pathogenesis.

It is true that Arnold was right when he contended that the transitional stages between the various oral parasites did not necessarily support the unitarian idea of pathogenesis; however, it cannot be denied that such stages are consistent with this idea; if there were no transitions, the unitarian idea had to be refused.

As to the question whether our parasites should be regarded as double monsters (Ahlfeld), or whether they should be explained by dislocation (Schwalbe), it is significant that all encranii and pharyngeal and oral parasites were found to arise at or near the midline of the autosome. If dislocation were the right explanation, one should expect to find similar malformations elsewhere in the head, because its complicated development should predispose to dislocation in many different places.

The pathogenesis of the double monsters in general cannot be discussed here; but it is indicated to mention that they are now explained by the organizer theory of Spemann. Thus Krafka (1936) has postulated that these malforma-

tions arise through the production of secondary embryonic axes in the primitive shield as a result of interference with the normal effects of the organizers, their greater frequency in the upper and lower pole of the body being well explained as a function of the proximity of the potential fields of Hensen's node. It is obvious that this theory is consistent with the various facts which were discussed in this presentation. It also explains the axial orientation of the parasite, noted already by Ahlfeld, and so distinctly displayed in our case, namely, that the upper portion of the parasite contained head material, while the lower portion revealed extremities.

SUMMARY

The teratoid parasites of the mouth show all transitions from dermoids (hairy polyps) through teratomas (unorganized derivatives of all three germinal layers) to incompletely developed twins (sometimes affixed to the autosite by an umbilical cord). Most oral parasites are attached to the sphenoid bone (episphenoids); some are affixed to the palate (epipalati or epurani); a few are bound to the jaw (epignathi). The episphenoids mostly grow into the pharynx and mouth; in a considerable number of cases they develop into the cranium (encranii); in rare cases they extend into both directions. A case of the latter variety has been described in this paper. It has been shown that the intraoral and intracranial portions of these parasites are connected with one another through the area of the craniopharyngeal canal. Since many encranii spring from the region of the pituitary gland, and most oral parasites arise at or near the sphenoid bone, and since almost all encranii, episphenoids, epipalati, and epignathi develop at or near the midline, it appears that all these parasites have a common pathogenesis. As the higher-developed parasites, like the one reported here, show the same axial orientation as their autosites, it suggests itself that they are asymmetrical double monsters rather than teratomas due to dislocation of germ tissue.

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ANTI-INFECTIOUS PROPERTIES OF SALIVA—A REVIEW

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THE word "anti-infectious" in the title has a broader connotation than "antibacterial." An anti-infectious property is one that in some way might interfere with the consummation of infection by a pathogen. Antibacterial properties constitute one subdivision of anti-infectious properties. The possible anti-infectious effects of saliva, due to its physical or mechanical properties (e.g., as a diluent, as a demulcent over the oral mucosa and the teeth, its viscosity, the flushing effect due to its flow), are not considered in this review; nor are we considering possible antibiotic effects of the oral flora and fauna—setting an upper limit to their own numbers, maintaining the relative proportions of their several species, or preventing the establishment of potentially pathogenic intruders.

EFFECTS ON THE BACTERIAL CELL AND VIRUSES

Saliva in vitro can produce a number of demonstrable effects upon a variety of bacteria. These effects, however, are relatively mild compared with the effects of blood serum.

An antibacterial effect can be shown against, for example, *Pseudomonas aeruginosa*, a pyogen of secondary importance. Four dilutions of a 24-hour, 37° C. bouillon culture of this organism were prepared (Dilution 7-Dilution 10), ranging from 1 part in 10 million to 1 part in 10 billion. The diluent was sterile physiologic NaCl solution to which had been added a little nutrient gelatin (to protect the bacteria from the toxic effect of NaCl). Into each of four sterile tubes, was placed 1 c.c. sterile bouillon; into each of another set of four sterile tubes, was placed 1 c.c. of sterile physiologic NaCl solution; and into each of a third set of four sterile tubes, was placed 1 c.c. of fresh, unstimulated human saliva. *Ps. aeruginosa*

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did not develop in cultures taken from another portion of the saliva which was used in these tests. One-tenth cubic centimeter of Dilution 8 of *Ps. aeruginosa* was placed into one bouillon tube, one saline tube, and one saliva tube. One-tenth cubic centimeter of Dilutions 8, 9, and 10 were similarly placed into their respective tubes. All tubes were incubated at 37° C. After 3, 6, 9, and 23 hours, 1 loopful was taken from each of the 12 tubes and transferred to a tube of sterile bouillon. These subinoculations were incubated and examined for the presence of *Ps. aeruginosa*. The results are given in Table I. The pH of each of the 12 tubes was colorimetrically determined immediately after the last (23-hour) subinoculations had been made.

TABLE I

| INOCULUM | BOUILLON | | | | | 0.85 PER CENT NaCl | | | | | SALIVA | | | | |
|----------|----------|---|---|---|-----|--------------------|---|---|---|-----|--------|---|---|---|-----|
| | a | b | c | d | pH | a | b | c | d | pH | a | b | c | d | pH |
| Dil. 7 | + | + | + | + | 8.2 | + | + | + | + | 8.0 | 0 | + | + | + | 7.7 |
| Dil. 8 | 0 | + | + | + | 8.3 | + | + | + | + | 8.3 | 0 | 0 | 0 | + | 7.8 |
| Dil. 9 | 0 | + | + | + | 8.3 | 0 | + | + | + | 8.4 | 0 | 0 | 0 | 0 | 7.8 |
| Dil. 10 | 0 | + | + | + | 8.3 | 0 | 0 | 0 | 0 | 8.4 | 0 | 0 | 0 | 0 | 7.9 |

a, 3 hours; b, 6 hours; c, 9 hours; d, 23 hours.

+, growth of *Ps. aeruginosa*. 0, no growth. The number, expressing the dilution of the culture, used as inoculum, is the negative exponent to the base 10. E.g., Dil. 7 means that 1 part of culture was added to 9,999,999 parts of sterile diluent; Dil. 8 means that 1 part of culture was added to 99,999,999 parts of sterile diluent, etc.

In the bouillon tubes, inoculated with Dilutions 8, 9, and 10, the number of organisms was so small that their increase during the first three hours of incubation was not enough for the samples, transferred at that time as subinocula, to yield growth. However, from the sixth hour on, the increase was sufficient for the subinoculum to establish growth. The same observation can be made in respect to the saline tube, inoculated with Dilution 9, and to the saliva tube, inoculated with Dilution 7. In the saliva tube, inoculated with Dilution 8, multiplication was so retarded that more than nine hours were required before the number of organisms had increased sufficiently to yield growth when 1 loopful was transferred to a tube of sterile bouillon.

Theoretically, 16 positive cultures might have been obtained from the bouillon tubes, the saline tubes, or the saliva tubes. The ratios of observed to theoretically possible positive cultures were, respectively, $\frac{13}{16}$, $\frac{11}{16}$, and $\frac{4}{16}$ (0.81, 0.69, and 0.25). Saline apparently has a slight toxicity for *Ps. aeruginosa*, which is revealed with small inocula (Dilution 10). In terms of the number of organisms, killed, or whose development is prevented, saliva under the conditions of the test is more than 10 times and less than 100 times as antibacterial as is saline. The effect here demonstrated can be due neither to lack of food (saline has even less of nutritive value than does saliva) nor to an unfavorable pH.

TABLE II

| 1 | 2 | 3 | 4 | 5 | 6 |
|--------|--------------------|----|--------------|-------|---|
| LIQUID | NUMBER OF CULTURES | | | | AVERAGE NUMBER OF DAYS AFTER INOCULATION BEFORE GROWTH APPEARED |
| | + | 0 | CONTAMINATED | TOTAL | |
| R | 41 | 4 | 1 | 46 | 28 |
| S | 0 | 45 | 1 | 46 | |
| SF | 1 | 12 | 0 | 13 | 40 |
| SH | 0 | 39 | 3 | 42 | |
| SFH | 3 | 9 | 1 | 13 | 42 |
| D | 9 | 1 | 1 | 11 | 32 |

+, growth of *M. tuberculosis* on Corper's medium.

0, no growth of *M. tuberculosis* on Corper's medium: recorded only after 110 days' incubation.

R, Ringer's solution, sterile.

S, fresh, unstimulated, centrifuged human saliva.

SF, same as S, filtered through Jena fritted glass filter G 5/3. No growth obtained from subcultures of SF.

SH, same as S, heated at 56° C. for 30 minutes.

SFH, same as SF, heated at 56° C. for 30 minutes.

D, distilled water, sterile.

A few years ago in my laboratory, Dr. Kanter and Mrs. Dietz studied the effect of human saliva on tubercle bacilli.¹⁵ One part (0.1 c.c.) of a suspension of human tubercle bacilli was suspended in 5 parts (0.5 c.c.) of several liquids. Samples (0.2 c.c.) of these mixtures were immediately inoculated upon Corper's medium.⁶ The results are summarized in Table II. The saliva used was obtained from four adults (pooled saliva was not used).

None had a history of clinically active tuberculosis. Two reacted strongly, locally and systemically, to the i.c. injection of 0.1 mg. of old tuberculin. Another was a nonreactor. Chest x-rays of the fourth were negative.

The four instances where viable tubercle bacilli were not obtained from the suspension in Ringer's solution, are probably attributable to the small number of organisms in the original suspension from which the inocula were taken. The results strongly indicate that the presence of saliva in the inoculum tends to prevent the growth of tubercle bacilli on Corper's medium. The same effect was observed when other media, on which tubercle bacilli will grow, were substituted for Corper's. The effect was still demonstrable when saliva was diluted with 4 parts of sterile Ringer's solution. The antibacterial property is not due (1) to the bacteria of the saliva (SF and SFH), although the role of filtrable products of bacterial metabolism was not eliminated, (2) to a complement-amboceptor mechanism (SH and SFH), or (3) to the hypotonicity of saliva (D). Concentrations of sulfoneyanid, within the range of concentrations of this salt in saliva, do not prevent the growth of tubercle bacilli. This last observation is contrary to an earlier report.²⁰

The data given in column 6, Table II, suggest that, even in those cases in which tubercle bacilli were recovered from saliva suspensions (SF, SFH), there was operative a bacteriostatic effect. The number of such cases, however, is too few to justify such an inference.

Centrifuged saliva suspension of tubercle bacilli, injected subcutaneously into guinea pigs, immediately after mixing (2 animals) and after two hours' incubation at 37° C. (2 animals), produced tuberculosis. Filtered, centrifuged saliva suspensions of tubercle bacilli, injected subcutaneously into guinea pigs immediately after mixing (3 animals) and after two hours' incubation at 37° C. (5 animals), produced tuberculosis. The inocula were not greater than, and in most cases probably less than, the inocula transferred to the Corper's medium in the *in vitro* tests. There was no significant difference between these experimental animals and control animals, receiving inocula of the same size, suspended in Ringer's solution, in respect to (1) time interval between injection and appearance of regional lymph node involvement, (2) rate of progress of the disease, (3) length of survival after injection, and (4) extent of involvement found at autopsy.

The results of the *in vitro* tests indicate that saliva has an antibacterial effect on tubercle bacilli. The results of the injections of tubercle bacilli, suspended in saliva, into guinea pigs, indicate that the effect is bacteriostatic, not bactericidal.

Many kinds of bacteria other than *Ps. aeruginosa* and *M. tuberculosis* are sensitive to antibacterial properties of saliva, at least *in vitro*.⁴ Lysozyme appears to be one of the factors involved. Some bacteria are agglutinated in saliva. This raises the question of the presence of serologic antibodies in saliva. Saliva contains only about one-fourteenth as much protein as does blood serum. To what extent the antibody-containing globulin fraction appears in saliva, I do not know. Judging the body's economy from the normal functioning of the kidney, it is likely that the protein-conserving mechanisms of the salivary glands are normally highly efficient. Although serum antibodies can rise to high levels after infection or artificial active immunization, they soon tend to drop to "normal" levels. Thus, there is little reason to expect much, if any, antibody in saliva.

Saliva, *in vitro* and given time, can agglutinate typhoid bacilli: but this does not mean that specific, serologic agglutinins are present. Bacteria can be agglutinated by many nonspecific mechanisms. In one case, before, during, and after a course of typhoid bacterin, the saliva did not "agglutinate" *Eberthella typhosa* in dilutions greater than one-half, remaining virtually stationary during and after the immunization. During the same time the agglutinating titer of the subject's blood serum rose from 1/20 to 1/320.

Even if a certain amount of serum-globulin antibody was normally secreted into the saliva, it is not improbable that the amount secreted would not be increased during a rise in serum antibody. Diphtheria antitoxin apparently is secreted into saliva. Are the antibacterial effects (e.g., agglutinating power) of

saliva affected in the same way as are the true serum antibodies by physical and chemical agencies? This question has not yet been answered satisfactorily. Can we demonstrate the phenomenon of specific absorption of agglutination and other antibacterial effects in saliva? Questions of this sort would have to be answered before we would be justified in asserting that some of the antibacterial properties are due to true serologic antibodies.

Another anti-infectious property of saliva has been recorded: the transformation of diphtheria bacilli and pneumococci from virulent to nonvirulent types.

The effects of saliva upon the formation or action of hemolysins, leucocidins, and other exotoxins, of fibrinolysins, and of "spreading" factors remain to be studied.

Such evidence as is available does not suggest that saliva possesses antiviral properties. The viruses upon which observations have been made are PR8 strain of influenza,¹⁰ poliomyelitis,¹⁴ rabies,⁹ and mumps.⁵ This field should be more systematically studied.

EFFECT ON COAGULATION OF BLOOD

The ability of saliva to accelerate blood coagulation⁸ has often been regarded as a factor in preventing wound infection and in facilitating healing.

EFFECT ON CAPILLARY PERMEABILITY¹⁷

An increase in capillary permeability, permitting a local concentration of antibody-containing blood plasma and phagocytic leucocytes at the site of irritation, would be potentially an anti-infectious phenomenon. Sterile human saliva, injected into the skin of a rabbit, results promptly in a local increase in capillary permeability. The factor or factors responsible are not the salts, proteins, enzymes, ether-soluble substances, mucin, or the hypotonicity of saliva. The effect is demonstrable when whole, stimulated, centrifuged saliva is diluted with sterile distilled water to one-twentieth of its original strength.

CHEMOTACTIC INFLUENCE UPON LEUCOCYTES⁷

Living leucocytes from the rabbit move in vitro toward human saliva. Fig. 1 summarizes a series of observations on which the above statement is based. Centrifuging the saliva, filtering it free of bacteria, or heating it (56° C. for thirty minutes or 70° C. for thirty minutes) lessened but by no means abolished the chemotactic effect. The hypotonicity of saliva is a negligible factor. The significance of this prochemotactic effect lies in the importance of leucocytes (particularly as phagocytes) in many pathologic processes.

EFFECT UPON PHAGOCYTOSIS

In a recent study¹² human leucocytes were used. The bacteria employed were a lactobacillus, a streptococcus, and *Sarcina lutea*. Leucocytes-bacteria-saliva mixtures showed more phagocytes than did leucocytes-bacteria-physiologic saline mixtures, but many fewer phagocytes than did leucocytes-bacteria-human serum mixtures. In fact, saliva seemed to depress the phagocytosis-promoting property of serum: an observation in harmony with an earlier one¹⁹ to the effect that salivary mucin inhibited phagocytosis of an alpha streptococcus and *Staphylococcus albus*.

The original report¹² does not give enough data to justify an opinion upon the significance of the differences in the degree of phagocytosis.

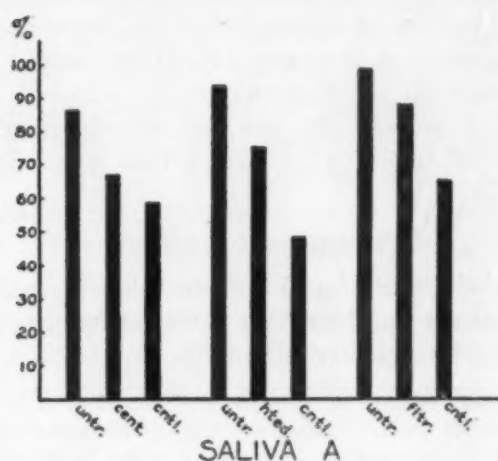


Fig. 1.—Percentage of leucocytes observed moving toward micropipet containing saliva as collected (untr.), saliva which had been centrifuged at 3,000 r.p.m. for ten minutes to remove some of its bacteria, debris, salivary corpuscles, and other particulate matter (cent.), liquid part of exudate from which leucocytes had been removed by centrifuging (cntl.), saliva which had been heated at 70° C. for thirty minutes (hted.), and saliva which had been freed of bacteria by filtration through a Jena fritted glass filter, G 5/3 (filtr.) (From Dietz: J. Dent. Research 18: 367, 1939.)

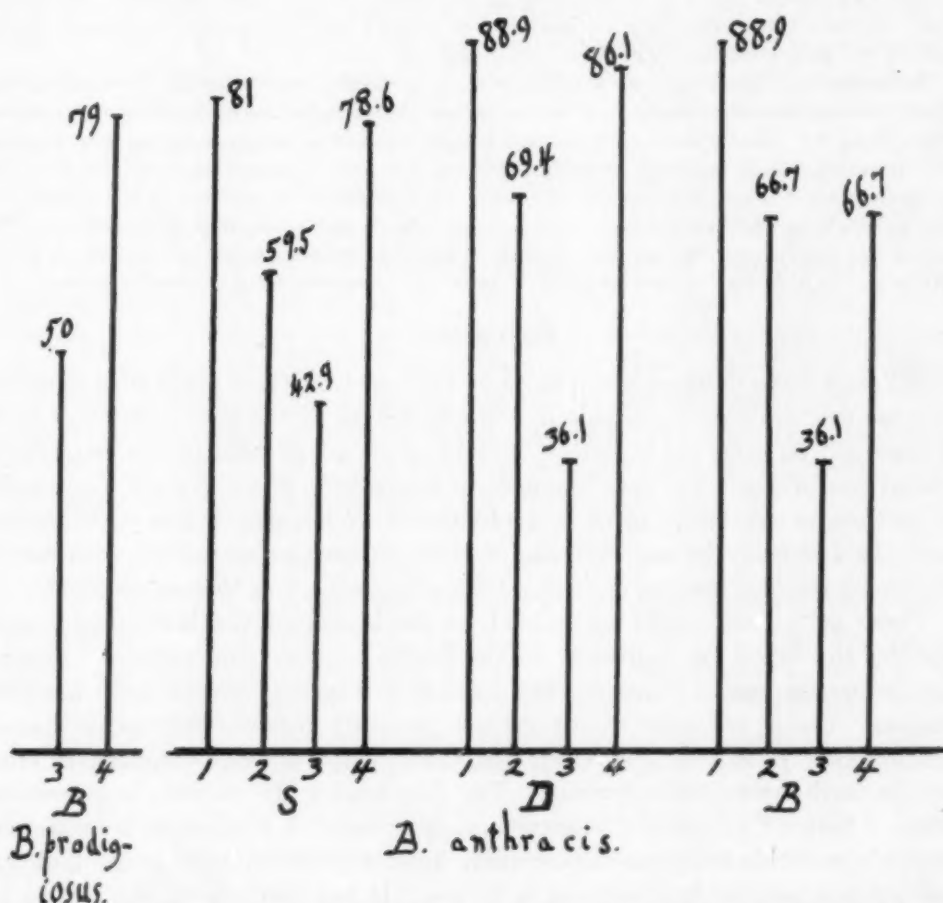


Fig. 2.—Percentage of white mice surviving intraperitoneal injection. Showing protective effect of centrifuged saliva. Capital letters under groups of vertical lines represent different individuals furnishing the saliva. Arabic numeral under each vertical line represents group of mice, receiving treatment, described in text. Percentage of survivors based on results obtained in 100 mice, each for lines 3 and 4 (*B. prodigiosus*), in 42 mice for each of the other lines.

The factor or factors responsible for the purported greater degree of phagocytosis in the presence of saliva alone than in saline alone, have not yet been identified. The assertion that saliva contains opsonin is premature. This assertion rested in part on the fact that saliva, exposed to bacteria, lost its phagocytosis-promoting power. It was not demonstrated that bacteria, so treated, were more susceptible to phagocytosis than were bacteria which had not been exposed to saliva.

MOUSE-PROTECTIVE EFFECT^{1, 2}

Unstimulated, whole, centrifuged, human saliva increases the resistance of young adult white mice to at least two bacterial species (*B. prodigiosus* and *B. anthracis*), injected intraperitoneally (Fig. 2).

B. prodigiosus. Equal volumes of same culture diluted to same extent (a) with sterile physiologic saline, (b) with saliva. Inoculum 0.5 c.c. intraperitoneally, respectively, into Groups 3 and 4. Group 4, receiving the bacteria suspended in saliva, showed a significantly greater proportion of survivors, 72 hours after inoculation. *B. prodigiosus* was recovered in heart blood cultures from the survivors, autopsied after 72 hours, more frequently from Group 3 than from Group 4 (saliva). Among the animals dying within the first 72 hours after injection, death came earlier to animals in Group 3 than to animals in Group 4 (saliva).

B. anthracis. Young, adult mice were selected and divided into 4 groups. Group 1 received intraperitoneally 0.5 c.c. of saliva. Group 2 received subcutaneously 0.5 c.c. of saliva. The other two groups received no injections at this time. Twenty-four hours later, Groups 1, 2, and 3 received intraperitoneally an inoculum of anthrax bacilli suspended in saline. Group 4 received the same-sized inoculum suspended in saliva.

Referring to Fig. 2, we see that the greatest protection was afforded when saliva was injected intraperitoneally twenty-four hours before the intraperitoneal injection of anthrax bacilli (Group 1). Saliva from individuals S and D, injected at the same time as the anthrax bacilli, provided almost as good protection (Group 4). Some protection is afforded by the saliva even when it is not injected into the same site as receives the anthrax bacilli (Group 2). Group 3, which at no time received saliva, showed the lowest percentage of survivors. The inoculum was intentionally adjusted so that there would be an appreciable number of survivors in Group 3. In this way it became possible to reveal relatively small protective power.

DISCUSSION

We shall have to know much more of the anti-infectious properties of saliva before we can evaluate their role in the physiology of the oral cavity in health and disease. To what are they due, individual chemical constituents or physico-chemical conditions? Do they fluctuate in intensity? If so, is there a recognizable pattern or regularity in their fluctuations? What determines such fluctuations? Is deficiency in one or more of these properties correlated with actual infection of the oral mucosa or with a lowered resistance of this membrane?

These properties might be related to the health of the individual manufacturing the saliva or indirectly to the health of other individuals. Though these properties are of relatively low intensity, they may be adequate for most occasions. Under ordinary conditions one probably receives only small inocula of pathogens. It is widely believed that saliva helps to keep wounds free from infection and favors their healing. The dog licking its wounds is a familiar picture. Miller¹⁸ repeatedly observed phagocytosis in extraction wounds and thought it probable that this phenomenon materially contributed to the fact that these wounds usually heal without infection. It has been shown that saliva attracts leucocytes, but it has also been shown that it depresses phagocytosis. The salivary glands were removed from a dog and then a wound was inflicted on the oral mucosa. In another dog, only the wound was inflicted. Bacteria were present in the wounds of both dogs. Healing proceeded smoothly in both wounds and there were no complications.¹¹

Bacteria of the oral cavity are involved in gingivitis and stomatitis. Does the bacteriostatic effect of saliva on tubercle bacilli account at least in part for the rarity of tuberculous lesions of the oral mucosa? What are the relations between the oral flora and infections of the salivary glands and their ducts? Do low anti-infectious properties of saliva favor infections of these structures? To what degree is the oral flora responsible for infections of the tonsils, the oropharynx, the nasopharynx, the nasal cavity, and the deeper levels of the respiratory system? The saliva is the most important single factor in influencing the bacterial content of the stomach.¹⁶ The oral cavity seems to be an important reservoir from which the lactobacilli of the intestine are supplied.¹³ Does the oral cavity supply other kinds of bacteria to the intestine? The ameba and trichomonad of the oral cavity are specifically different from the forms found in the intestine. Factors, such as the antibacterial properties of saliva, which influence the kinds and numbers of mouth bacteria, may have to be taken into account in reaching anything like complete answers to the above questions.

In talking, sneezing, and coughing, droplets of saliva are ejected into the air.³ These droplets contain microorganisms important in air-borne infections. These air-borne infectious agents come from the oral mucosa as well as from various levels of the respiratory tract: a fact often unknown or ignored. How does saliva affect the kinds and numbers of infectious agents thus ejected, their survival in the air and dust, and their communicability and infectiousness after reaching de-epithelized wounds and the oral and respiratory mucosae of the recipient? These are important questions for the mechanisms and epidemiology of air-borne infections.

SUMMARY

Saliva has a number of properties, any one of which might make it a little more difficult for a pathogen to establish an infection: bacteriostatic, bactericidal, agglutinative, transformative or mutative (from a virulent to a non-virulent type), accelerating blood coagulation, attracting leucocytes, increasing capillary permeability and "mouse-protective." The possibility is pointed out that these properties may influence (a) the occurrence, severity, and progress of infections of oral, pharyngeal, nasal, and respiratory mucosae, and salivary glands and ducts, (b) the bacterial flora of the stomach and intestine, and (c) the epidemiology and communicability of air-borne infections.

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TWO CASES OF SUBLINGUAL DERMOID AND A CASE OF PROTRUSION OF MANDIBLE

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SUBLINGUAL DERMOID

DERMOIDS in the region of the floor of the mouth are congenital inclusions of epidermis within the deeper tissues. They may not be evident at birth, and may not be noticed until later in life. The dermoid consists of a fibrous capsule lined with squamous epithelium, containing a substance that may be watery or milky in consistency or may be a thick greasy paste. Hair or other skin appendages may be present. According to Butlin in his excellent book on *Diseases of the Tongue*,‡ dermoids, in relation to the floor of the mouth, occur

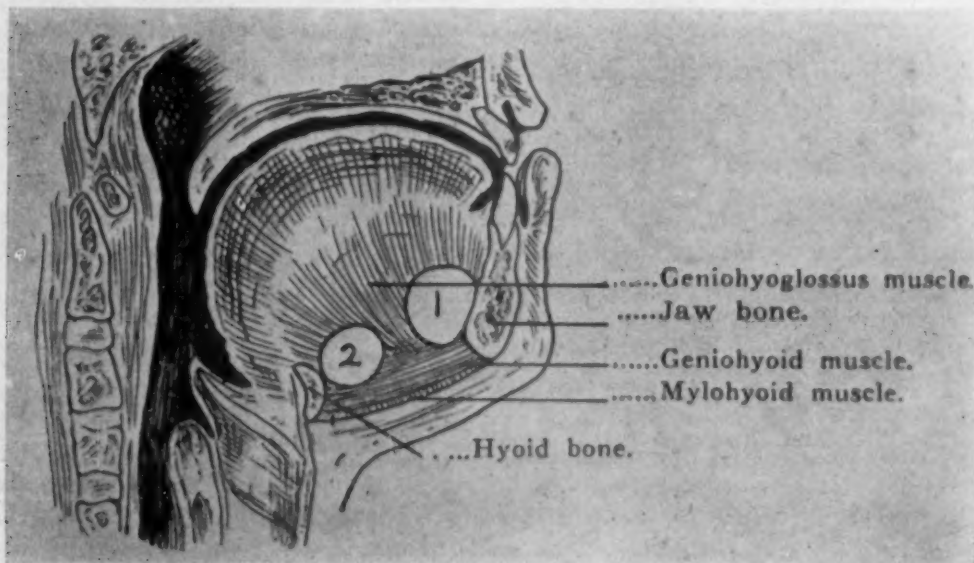


Fig. 1.—Diagram showing the location of median dermoids in the floor of the mouth. (From Butlin.)

Case reports from the Departments of Plastic and Maxillo-Facial Surgery, Graduate Hospital and Oral Surgery Clinic, School of Dentistry, University of Pennsylvania.

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‡Butlin, H. T.: London, 1885, Cassell & Co., p. 238.

in one of two situations: in the midline beneath the skin or between the geniohyoglossus muscles; or laterally below the angle of the jaw. Butlin is of the opinion that laterally situated dermoids, which do not represent the remains of a branchial pouch, were once median and have shifted their position during development. Fig. 1 is from Butlin and shows the location of median dermoids of



Fig. 2.—Case 1. Sublingual dermoid, profile view showing swelling beneath chin.



Fig. 3.—Case 1. Same as Fig. 2, front view.

the floor of the mouth. When small, dermoids usually protrude downward beneath the chin, but may bulge upward into the floor of the mouth, in which case they appear as a yellowish mass beneath the mucous membrane. When very large, a dermoid may press the tongue upward and backward, and even cause dyspnea. A dermoid is to be distinguished from a ranula, which is entirely

above the floor of the mouth and therefore does not cause a swelling beneath the chin, has a bluish translucent appearance, is always unilateral at first, and when sufficiently large gives a distinct sense of fluctuation. The ranula contains material like the uncooked white of an egg. On bimanual palpation, dermoids yield a doughy sensation. A dermoid, situated deep in the muscles near the hyoid bone, might be difficult to distinguish from a thyroid tumor or cyst in this situation. However, the dermoid is practically never below the level of the hyoid bone, while the thyroglossal cyst is hardly ever above it.

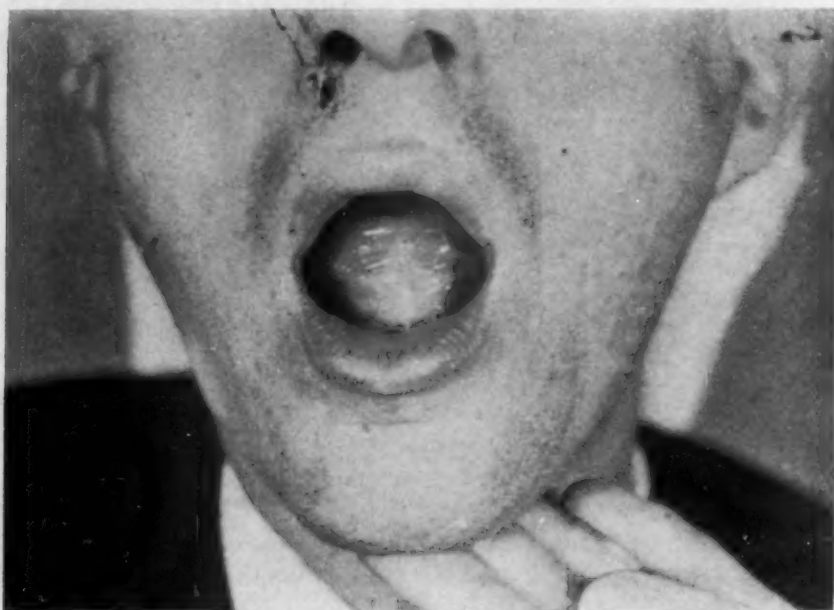


Fig. 4.—Case 1. Bulging of cyst beneath mucosa of floor of mouth.



Fig. 5.—Case 1. Sublingual dermoid after removal. Note sebaceous material filling cyst cavity.

The treatment of sublingual dermoid is complete enucleation. If the mass is situated immediately beneath the mucous membrane, it is possible that it can be removed from within the mouth by a free incision and blunt dissection, but usually it is preferable to approach it from below. It is absolutely necessary to remove the whole epithelial wall. When approached externally, per-

fectly free access and a clear view of the tumor can be obtained. Furthermore, the wound can be kept aseptic, the bleeding is easily controlled, and a properly placed skin incision will leave no disfiguring scar.

The following two cases are typical examples of this condition:

CASE 1.—C. W. S., a man aged 36 years, was first seen in May, 1941, presenting a painless, soft, smooth doughy mass in the midline beneath the chin (Figs. 2 and 3), which gave him little or no inconvenience except the disfigurement, and which he thought was slowly growing larger although it had been noticed for about eight years. On pressure from below, the mass was found to bulge up beneath the mucous membrane of the floor of the mouth (Fig. 4). On June 6, 1941, under general anesthesia, a transverse skin incision was made beneath the chin, exposing the mass, which was found to be encapsulated and lying between the geniohyoglossus muscles. The thick capsule was easily separated by blunt dissection from the surrounding soft tissues, and the entire cyst removed in one piece without rupture (Fig. 5). The wound was closed in layers, without drainage. Healing was uneventful.

Pathologic Report.—The specimen consists of a thin-walled, tough, brownish-gray cyst, filled with greasy yellowish-white amorphous material weighing in all 30 grams. Microscopically, the cyst wall consists of partially hyalinized thick connective tissue, imbedded in which are clusters of sebaceous glands. The lining of the cyst consists of stratified squamous epithelium overlaid with small amounts of debris.



Fig. 6.—Case 2. Sublingual dermoid, showing swelling beneath chin.

CASE 2.—E. McG., a man aged 21 years, was first seen on May 28, 1943. For several years he had noticed a painless swelling beneath the chin and in the floor of the mouth, but this gave him surprisingly little inconvenience, and he only sought treatment when rejected for military service on account of the swelling. The swelling was visible in the midline beneath the skin of the chin, and felt like a smooth, doughy mass (Fig. 6). On inspection of the mouth, at first the tongue was not visible, being pressed backward by the smooth mass beneath the floor of the mouth. When asked to protrude the tongue, only the tip could be seen, giving the appearance shown in Fig. 7. For the past two or

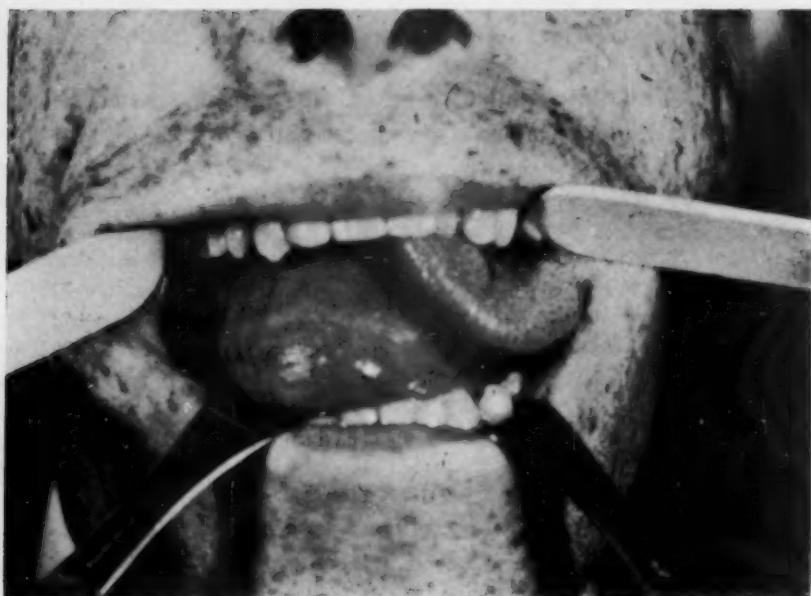


Fig. 7.—Case 2. Showing smooth mass in floor of mouth, preventing protrusion of tongue.



Fig. 8.—Case 2. Showing separation of incisor teeth due to pressure of mass in floor of mouth.

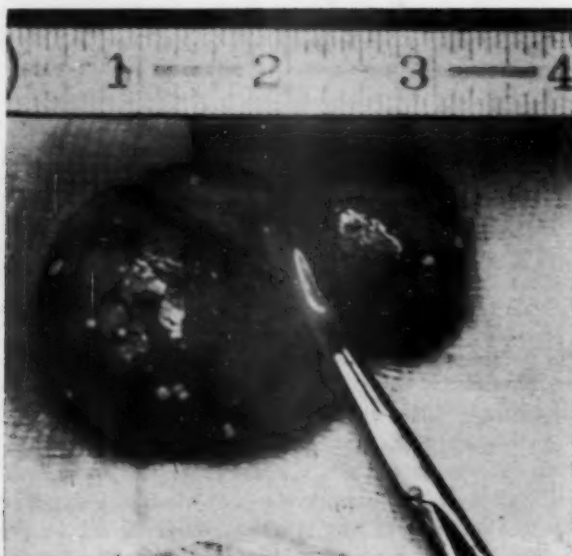


Fig. 9.—Case 2. Specimen after removal. Note hourglass shape.

three years he had noticed a gradual separation of the upper and lower incisor teeth (Fig. 8). This was due to slow pressure on the jaws by the mass. The swelling caused surprisingly little difficulty in speech and swallowing.

On June 6, 1943, under intratracheal anesthesia, the cystic sac was completely removed through a wide transverse skin incision beneath the chin. The sac was hourglass in shape (Fig. 9), one part being below and the other above the muscles of the floor of the mouth. A small rupture occurred at the constricted portion during removal. The sac contained thin milky fluid and floccules of cholesterolin, cheesy matter, and hairs. The separated muscles were brought together with catgut sutures and the skin incision was closed with silk. A rubber-dam drain was inserted. Healing was uneventful.

Pathologic Report.—The specimen consists of two adjoining thin-walled cysts, 4 cm. and 5 cm., respectively, in diameter. Each cyst is filled with numerous small smooth-surfaced yellowish globules of soft mushy material with many intertwining strands of hair. Microscopically, the cyst wall is composed of loose fibrocollagenous material in which are embedded several islands of sebaceous glands and sweat glands. The lining of the cyst is stratified squamous epithelium which in some areas is somewhat hyperplastic and shows occasional prickle cells. No distinct hair follicles are found in these sections, though hair is seen grossly in the lumen of the cyst. There is nothing to suggest neoplastic quality of cell change in any of these sections.

PROTRUSION OF MANDIBLE

G. K., a man aged 25 years, was first seen at Clinic at Evans Institute, March 20, 1944. He was discharged from the Army on account of a deformity of the jaw, which has existed as long as he can remember.



Fig. 10—Case 3. Protrusion of mandible, preoperative.

Examination showed a marked protrusion of the mandible, the lower anterior teeth being about half an inch in front of the uppers, giving him a very unusual facial contour (Fig. 10). There was practically no occlusal contact at all. Several molar and premolar teeth were missing in each jaw, but one molar was present above and below on each side. Study casts of the teeth were made, and it was found that a satisfactory occlusion of the remaining molars could not be obtained by sliding the entire mandible back. It was found, however, by taking out a segment of the edentulous part of the lower cast on each side,



Fig. 11.—Case 3. Relationship of upper and lower teeth, preoperative.

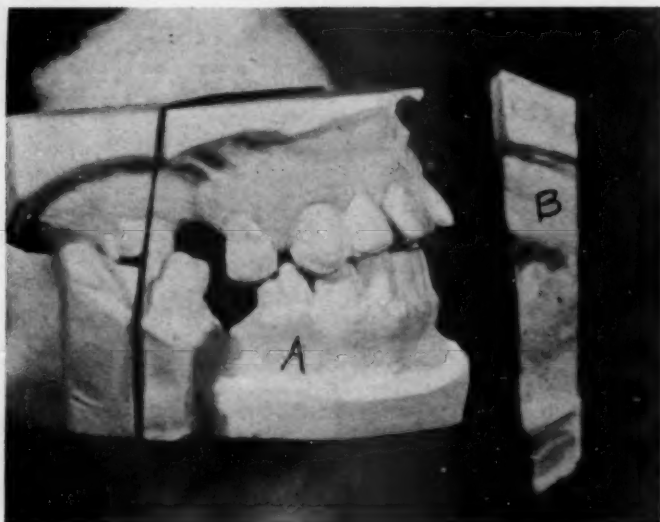


Fig. 12.—Case 3. A, Relationship of teeth on casts after removal of section from edentulous part of lower cast on each side. B, Section of cast removed.



Fig. 13.—Case 3. Arch wires applied for postoperative fixation.

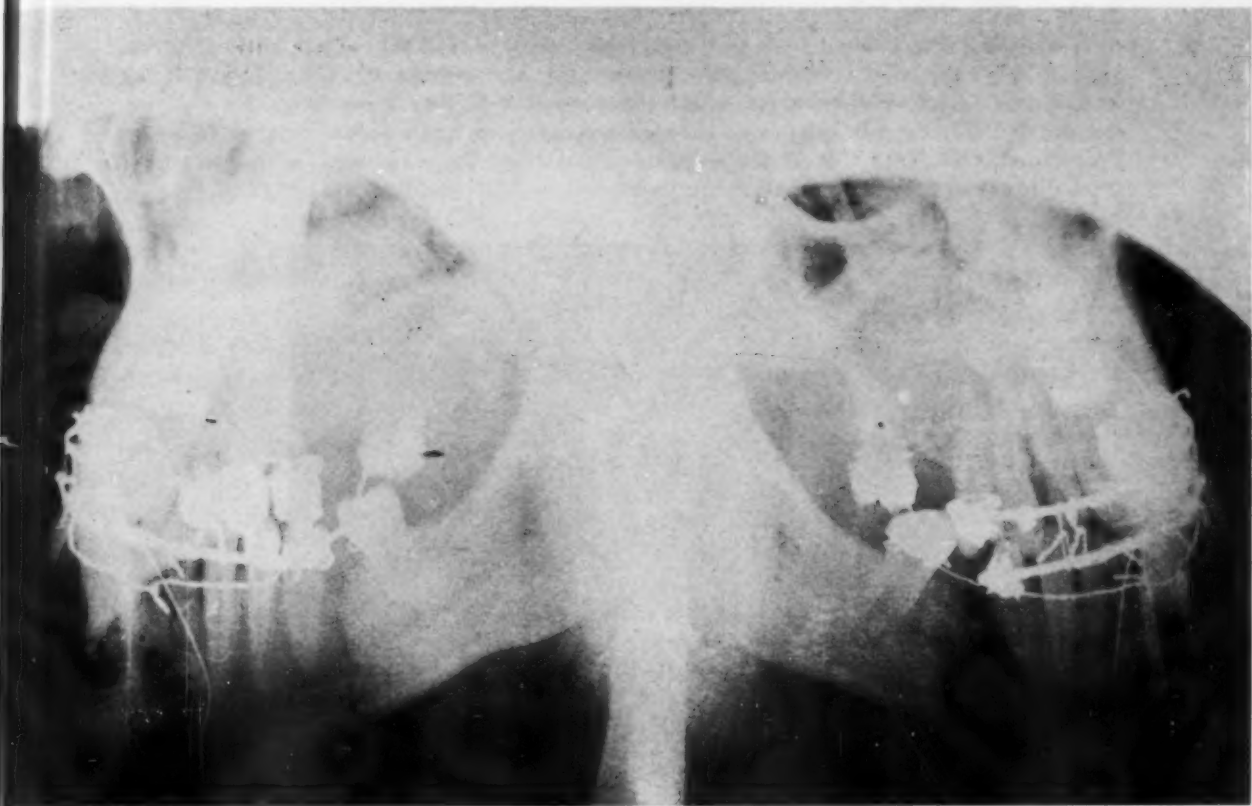


Fig. 14.—Case 3. Postoperative radiographs showing relationship of fragments.



Fig. 15.—Case 3. Front view after operation.



Fig. 16.—Case 3. Profile after operation.

that the molar teeth remained in fair occlusion, while the anterior segment of the mandible could then be brought back to give a satisfactory relationship of the anterior teeth. Fig. 11 shows the existing relationship of the upper and lower teeth. Fig. 12 shows the occlusion achieved by removal of segment from each side. As a result of this study it was decided to remove a section of bone, corresponding to the amount removed from the plaster cast, from each side of the edentulous portion of the mandible. Arch wires were applied to the upper and lower teeth for postoperative fixation (Fig. 13).



Fig. 17.—Case 3. Showing ability to open mouth after operation.



Fig. 18.—Case 3. Postoperative occlusion.

On April 14, 1944, at Graduate Hospital, the operation was performed under ether anesthesia. On each side a skin incision was made beneath the lower border of the edentulous portion of the mandible, the soft tissues were freed from the bone, and a section of bone, about $\frac{1}{2}$ inch in length, was removed from each side with a Gigli saw. This permitted the chin segment to be set back, after which the teeth were wired in occlusion. The skin incisions were closed with silk sutures, small rubber drains being left

in for a few days. Union of the fragments was very slow, a period of about six months elapsing before the jaw was sufficiently solid to permit removal of the wires from the teeth. Fig. 14 is made from postoperative radiographs, showing the position of removal of the sections of bone. Complete union eventually occurred, with marked improvement in function of the jaw and appearance of the patient. Fig. 15 is a front view and Fig. 16 a profile view of the patient after operation, while Fig. 17 shows the extent of mouth opening postoperatively. Fig. 18 shows rather imperfect but greatly improved occlusion of the anterior teeth.

For the majority of cases of mandibular protrusion we greatly prefer another operation than the one just described, namely, a horizontal or slightly oblique section high up through the ascending ramus on each side, and sliding the remainder of the mandible back on these cuts until the teeth can be fastened in occlusion. By this procedure only one cut through the bone is required on each side, no bone is removed, no teeth are sacrificed, the mouth is not entered, consequently the wounds remain clean, and, in most cases the cut being made above the mandibular foramen, there is no injury to the inferior dental nerve or vessels. The scars from the operation are inconspicuous. Union of the sectioned fragments is usually much more rapid than in the case of removal of a section from the body of the bone. Removal of a section from the body of the bone on each side is reserved for cases where teeth are missing at the point of section and where a more satisfactory occlusion can be obtained than by sliding back on the ascending rami.

AN ANALYSIS OF THE MECHANICS OF THE PERIODONTAL MEMBRANE

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THE problem of the part played by the periodontal membrane in supporting the tooth in its socket under masticatory load has always been one of interest to the dental profession, in the past largely academically, but now with increasing practical importance. It has long been conceded that the overstressing of this tissue plays an important part in periodontal disease. The question therefore arises as to what constitutes overstressing. This calls for an understanding of the mechanical problems involved in tooth support.

The generally accepted theory is that the tooth is suspended in its socket by the fibers of the periodontal membrane, one end of which is attached to the lamina dura of the socket and the other to the cementum of the root. When the tooth is loaded these fibers are put under tension.

There are several considerations which tend to nullify this simple explanation of tooth support. In the rest position these fibers are not under tension and so are exerting no force. For them to exert a supporting force they must be under tension, which requires an elongation of the fibers, and this in turn implies movement of the tooth. Since many of these fibers are wavy, a certain amount of the tooth movement would be necessary to straighten them before they would be put under any tension. Also, since they are not parallel to the

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direction of movement of the tooth, only a portion of the stress in them is effective, and therefore the total tension on the fibers would be much greater than if the forces were more advantageously applied. From these considerations it could be expected that the tooth would undergo considerable movement under load, which in the normal healthy case it does not do.

This oversimplification of the problem also assumes that the fibers are free, with no solid or liquid filling the spaces between them. But tissue fills the spaces between the fibers and this tissue is composed largely of fluid apart from that in the blood circulatory system. The fluid cannot readily pass from one part of this space to another because it is impeded by the solid material also occupying the space. When the tooth is depressed in the socket, the space between the root and the wall of the socket tends to decrease and its contents are put under pressure. Being practically incompressible, however, the only possible way for space to be provided for the root to move into is for the membrane to be squeezed away from the points of higher pressure (the decreasing space) toward those of lower pressure (where more space is provided). Since the surfaces of the membrane are attached to the root and its socket, they are relatively fixed so that only the tissue between these surfaces is displaced. The membrane is, therefore, subjected to a shear. The amount of movement of the tooth would be in proportion to the amount of this shear and therefore inversely proportional to the membrane's resistance to shear. The same principle applies, of course, when a transverse instead of an axial load is applied to the tooth.

Before proceeding, a few mechanical principles should be pointed out. When any material is subjected to an external force, it undergoes a change of size or a change of shape. This change is known as a strain. If the material possesses the property of returning to its original size or shape, it is said to be elastic. The forces within the body of a material exerted by one part of it on another and tending to return it to its original size or shape are known as stresses. The stress divided by the strain is a measure of the elasticity of the body and is known as the elastic modulus. This may just as conveniently be considered as a measure of the resistance to change of size or shape. When the term modulus of elasticity or Young's modulus is used, it applies to tensile and compressive stresses and strains.

The modulus which applies to shear stresses and strains is known as rigidity. To illustrate rigidity (Fig. 1) we can consider a cube of material fastened to a rigid surface. If a tangential force is applied to the top surface of the cube, then the cube would become a rhomb. The amount of horizontal displacement of the top surface of the cube divided by the thickness of the cube is a measure of the shear strain. The total force applied tangential to the top surface of the cube divided by the area of the surface is a measure of the shear stress. The rigidity is the shear stress divided by the shear strain.

It should be noted that in tension and compression the stress and strain measured are perpendicular to a transverse plane, while in shear they are parallel to it. Further, it can be shown that even under purely compressive or tensile applied forces there will be shearing stresses in any plane neither perpendicular nor parallel to the applied force. This shearing stress increases to a maximum at 45 degrees, and then decreases to zero at 90 degrees (Fig. 2).

In the diagram, the force P is resolved at the chosen plane into two components at right angles to each other. The component, S , is the projection of P on the plane and increases with the angle θ . The area of mn , A' , increases less rapidly than S up to 45 degrees and more rapidly thereafter, so that the

shear stress, $S_s (= S/A')$ increases up to 45 degrees and decreases thereafter. S is 0 at 0 degrees and A' is infinity at 90 degrees so that S_s is 0 at both of these angles.

If the material is subjected to a compressive force but is incompressible, it could be strained only in shear. This is the case with the periodontal membrane when considered incompressible, as has already been shown. Actually, a liquid such as water is not entirely incompressible but has what is known as a high bulk modulus, which means that a large pressure is required to produce a very small fractional decrease in volume.

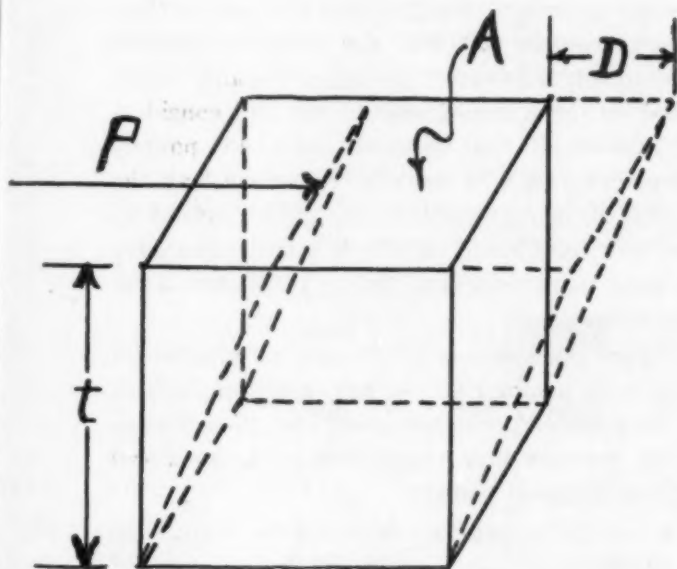


Fig. 1.

Fig. 1.—Rigidity— A = area of top; D = displacement; t = thickness. Shear strain $= D/t$; shear stress $= P/A$; rigidity $=$ shear stress \div shear strain.

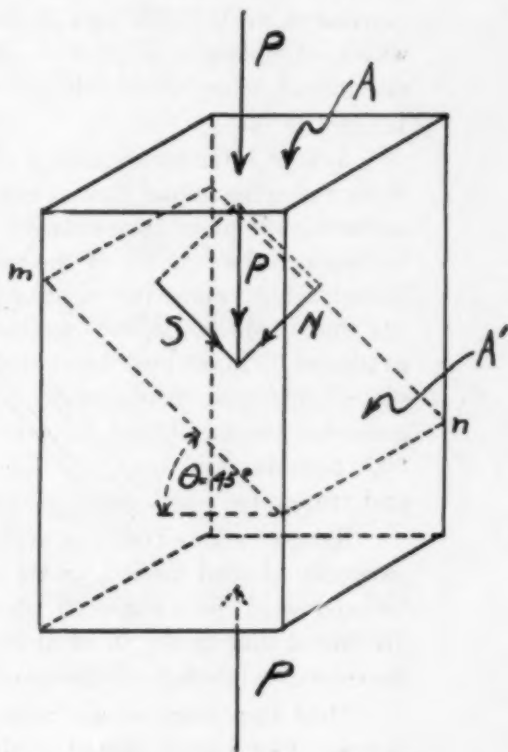


Fig. 2.

Fig. 2.—Shear stress on 45-degree plane. A = area of face; P = applied force; S = shear force; N = normal (perpendicular) force; A' = area of mn ; Compression stress $= C_s$; shear stress $= S_s$. $C_s = P/A$; $S_s = S/A' = 1/2 P/A$.

The physical concept just presented and the mathematical analysis of the mechanics involved in the periodontal membrane were first presented by J. L. Synge,¹⁰ Professor of Applied Mathematics, University of Toronto, at the instigation of Box, and later by Hay. Synge's original work was previously reported by this writer.³ What follows is a résumé of the points of major interest to the dental profession brought out in their publications.

As has been indicated, what cast the chief doubt upon the fiber support theory is the small observed movement of the tooth under load. Synge¹⁰ pointed out that, according to the fiber theory of support, the movement of the tooth should be in proportion to the thickness of the periodontal membrane, and when there is an increase in thickness because of congestion or other pathologic change, the movement of the tooth should correspondingly increase. With the theory of the incompressible membrane, the movement of the tooth would vary as the cube of the thickness. For example, assume a normal membrane of average thickness and a movement of 1/1,000 inch under a given load. Under the old

theory if the membrane were doubled in thickness the movement would be 2/1,000 inch. Under Synge's theory, if the membrane were doubled the movement would be 8/1,000 inch; if tripled, 27/1,000 inch. As the movement of the healthy tooth is so small as to be scarcely observable, a congestion doubling the thickness of the membrane would still not give a very noticeable movement. However, multiplying the movement by 8 would make an appreciable difference. In the original investigation, Synge used 230 pounds per square inch, which is the rigidity of rubber, for the rigidity of the periodontal membrane and found that an axial load of over 1,300 pounds would be required to give an axial displacement of 1/1,000 inch if the membrane were able to withstand the stress which, of course, it could not. A transverse load of about 21 pounds applied at the incisal edge would give to the incisal edge a transverse displacement of 1/1,000 inch.

Synge⁸ later considered a compressible membrane and Hay⁴ made calculations using the actual figures expressing the compressibility of water rather than assuming the membrane entirely incompressible. Dymont and Synge¹ attempted to measure the rigidity of the periodontal membrane of young calves and lambs. Substituting values for rigidity based on these measurements but still considering the membrane incompressible, it was found⁴ that an axial load of 431 pounds produced 1/1,000 inch axial displacement and 6.78 pounds transverse load the same transverse displacement. Using the compressibility of water instead of assuming the membrane incompressible, values based on Hay's calculations gave 90.5 pounds axial load and 5.2 pounds transverse load for 1/1,000 inch axial and transverse displacements respectively.

Synge¹⁰ showed among many things the location of the axis of rotation of variously shaped models under differently applied forces, but, more important, he calculated the stresses set up in the periodontal membrane under these forces. He found that under an axial load the pressure was a maximum at the apex and decreased to atmospheric pressure at the gingival margin.

Most important of all, however, are the pressures calculated for transverse forces. For a cone-shaped model which most closely approximates the root of the incisor tooth there are two points of maximum and two points of minimum pressure and between these, points of atmospheric pressure. The pressure at the apex and at the alveolar crest is always atmospheric under a purely transverse load. The illustration (Fig. 3) from Synge's original paper shows the distribution of the pressure in the periodontal membrane for this model under a load of 19/100 pound applied at right angles to the axis of the tooth at the incisal edge. This load is sufficient to give zero pressure at point M₄ and is called the critical transverse load.

In mastication the pressures set up are transitory and are probably quite large. They would be determined in normal function by the discomfort caused, but might be sufficient in cases of too heavy transverse loads to damage the tissues in time. Because of the intermittent nature of the masticatory act, this momentary increase in pressure followed by its complete relief could act as a booster pump to the circulation in the periodontal membrane, thus giving weight to the clinical observation that a membrane that functions is healthier than one that does not. Also, since there would probably be a negative pressure in the capillaries upon relief of the pressure in the membrane because of its rigidity, it could be expected that microorganisms in the membrane would be drawn into the capillaries and expelled into the circulation upon the next application of pressure to the tooth. Fish² reported a transient bacteremia in patients with

a marked paradontosis upon chewing hard candy. Under conditions where the individual is under nervous tension and holds the teeth tightly together, pressures sufficient to interfere with the capillary blood supply might be maintained for long enough time seriously to injure the periodontal membrane. It would be interesting to know in this connection whether there was any increase in periodontal disease among men in the Armed Forces who saw actual battle service over those who did not, allowance being made, of course, for other factors which enter.

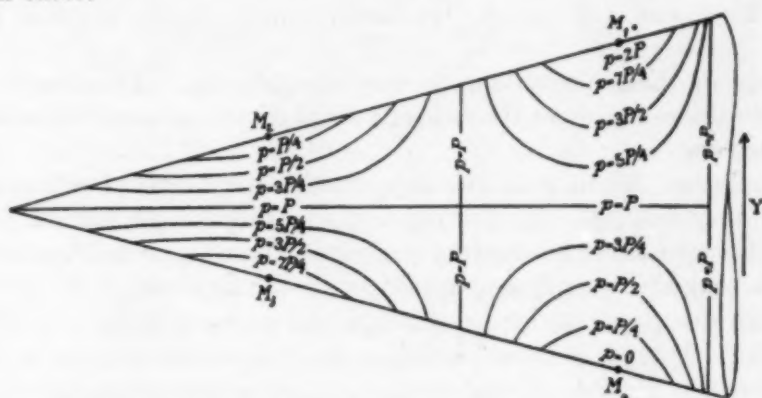


Fig. 3.—Distribution of pressure throughout the membrane of a cone-shaped model under a critical transverse force of 19/100 pounds applied at the incisal edge. P = atmospheric pressure; p = pressure in membrane. (From Syngé.¹⁰)

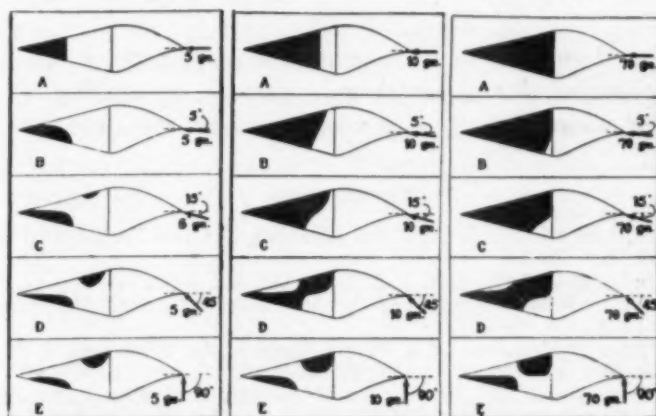


Fig. 4.—Areas of ischemia with loads of 5, 10, and 70 grams (0.18, 0.35, and 2.5 ounces, respectively) applied at various angles at the center of the incisal edge. (From Hay.⁵)

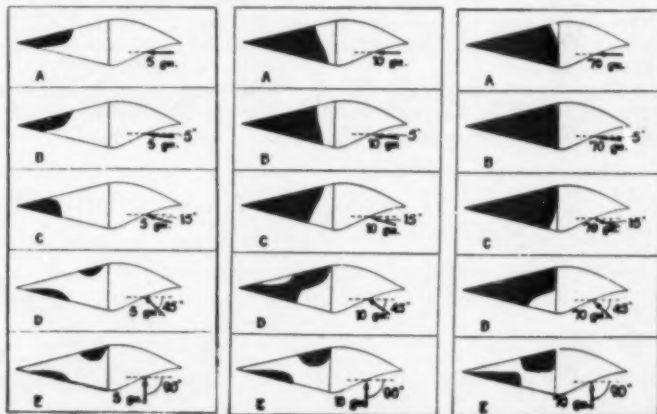


Fig. 5.—Areas of ischemia with loads of 5, 10, and 70 grams applied at various angles at the center of the lingual surface. (From Hay.⁵)

Hay⁵ has investigated the pressures set up in various portions of the membrane of a first maxillary incisor with a conical root when the force is applied at different angles at the incisal edge and at the lingual surface. To simplify the problem, it is assumed that the axis of root intersects the incisal edge.

The results of his investigation show that relatively small loads will produce ischemia of parts of the periodontal membrane. The illustrations (Figs. 4 and 5) taken from his article show the areas in which under the stated loads the pressures are sufficient to produce ischemia. The capillary blood pressure is taken as 23 grams per square centimeter which is the average given by Schwartz.⁷

A study of these illustrations is very enlightening. Attention is directed especially to the cases where the ischemic areas do not involve the membrane at the alveolar crest.

In conclusion, let us note the support the mathematical concept receives from laboratory findings. In addition to accounting for the clinically observed tightness of the tooth with a healthy periodontal membrane and the looseness of one with a congested membrane, a calculation of the location of the center of rotation (not discussed) under a transverse load agrees roughly with that found by Kronfeld.⁶ It must be remembered in this connection that the model under consideration was a right circular cone; also, that several other assumptions not mentioned here were made to simplify the mathematics. Further experiments performed by Schwartz with transverse loads on teeth of dogs showed "quite good" agreement with Hay's calculation of pressures that would cause strangulation.

Synge⁸ pointed out that of two methods of investigating this problem of the mechanism of the periodontal membrane, namely, by making a "model or by mathematics, the model is really much less powerful than the theoretical mathematical method."

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HEMATOLOGIC FINDINGS IN FUSOSPIROCHETAL STOMATITIS

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THERE are few data on the hematologic findings in patients with fusospirochetal stomatitis. Because of the similarity between the oral lesions of this disease and those associated with the blood dyscrasias, a blood count is frequently employed in making a differential diagnosis. It is important to know what variations in the hematologic findings, if any, occur in fusospirochetal stomatitis.

Bryant, in 1929, reported on the blood findings in 22 cases of Vincent's stomatitis. The total leucocyte counts were within normal limits. The absolute numbers of neutrophiles and lymphocytes were compared with normal values. The neutrophile counts in three cases were below normal values; the lymphocyte counts in 6 of the 22 cases were above normal values and one was below the normal value. These findings suggested that a relative lymphocytosis might be present in this disease.

More recently, Stine reported on the blood picture in 128 cases of Vincent's infection observed in a Student Health Service at a Midwestern university. It was not stated whether cases of Vincent's angina were also included in the series. There was no uniformity in the total or differential leucocyte counts. The total leucocyte counts ranged from 40,000 to 3,450 cells per cubic millimeter with the percentage neutrophiles varying from 93 to 12. Stine believed that the blood count was of no help in making a diagnosis and it may even be found confusing in that it suggests other diseases.

Tarnow reported on the hematologic findings in Vincent's angina. Slightly higher than normal values, including one count of 14,000 leucocytes per cubic millimeter were found. A slight leucocytosis might be expected in Vincent's angina where the deeper tissues of the throat are involved.

The blood counts analyzed in this report were obtained from patients receiving periodontal treatment in the Oral Medicine Clinic. The majority of the patients were male college students. The ages of the patients ranged from 12 to 37 years with most of the cases occurring under 25 years of age. There was a predominance of the male sex.

All of the patients were receiving treatment for fusospirochetal infection. The diagnosis of this disease was based on the presence of the usual clinical findings and corroborated by positive bacterial smear findings. The type of case ranged from mild, localized infections to acute generalized involvement of the oral tissues. Two patients had severe fusospirochetal ulcerations of the soft palate and cheek but no cases of true Vincent's angina were included in this group of patients.

The blood count was made at the first clinic visit, which as far as the duration of the disease was concerned, was not comparable in every patient. In many instances the onset of the acute fusospirochetal infection was preceded by

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an acute upper respiratory infection, or at other times an acute upper respiratory infection coexisted at the time the patients were under treatment in the Oral Medicine Clinic. On the basis of the clinical findings the cases included both acute and chronic fusospirochetal infections.

In addition to the usual differential blood counts made from the finger prick blood, differential leucocyte counts were also made from blood obtained from the gingiva at the site of the ulcerations. The finger prick blood was collected in the usual manner. The gingival blood specimen was obtained after the patient had rinsed his mouth with tap water and any accumulated debris had been mechanically removed. During the latter procedure sufficient bleeding occurred in the acute cases for making the smears for the differential leucocyte count. In chronic cases slight bleeding of the gingival margin was produced by means of a periodontal scaler.

The standard methods were employed for making the erythrocyte and leucocyte counts. All hemoglobin determinations were made on a photoelectric colorimeter. Two hundred cells were counted for all the differential leucocyte counts. In some cases, counts were made following the control of the disease. The results of the follow-up counts and the gingival differential counts will be analyzed in a future report.

DATA

The total leucocyte counts in the 110 cases of fusospirochetal stomatitis are shown in Fig. 1. The total leucocyte counts varied from a low of 2,900 (one case) to a high of 16,000 cells per cubic millimeter. If we accept the normal range of total leucocyte counts for adults of both sexes to be from 5,000 to 10,000 leucocytes per cubic millimeter as stated by Wintrobe, 78 per cent of the total leucocyte counts fell within this range of values. A study of the ages of the thirty patients having total leucocyte counts beyond the normal range given by Wintrobe showed that with four exceptions they were under 20 years of age.

Kolmer considers the range of total leucocyte counts in the age group 8 to 18 years to be from 4,500 to 13,500 cells per cubic millimeter. If these figures are accepted as the normal range of values, a still greater proportion of the leucocyte counts of patients with Vincent's infection would fall within the normal range.

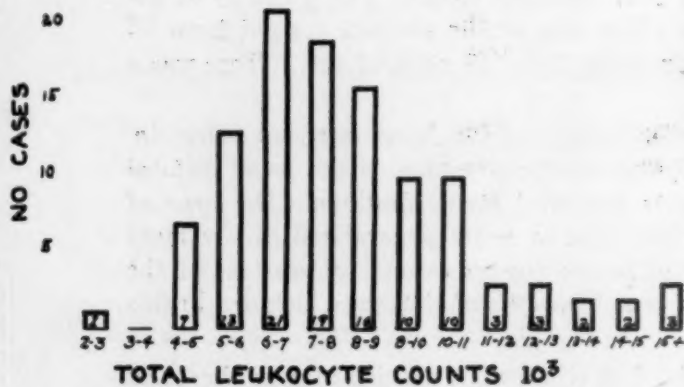


Fig. 1.

Fig. 1.—Distribution of total leucocyte counts in 110 patients with fusospirochetal stomatitis.

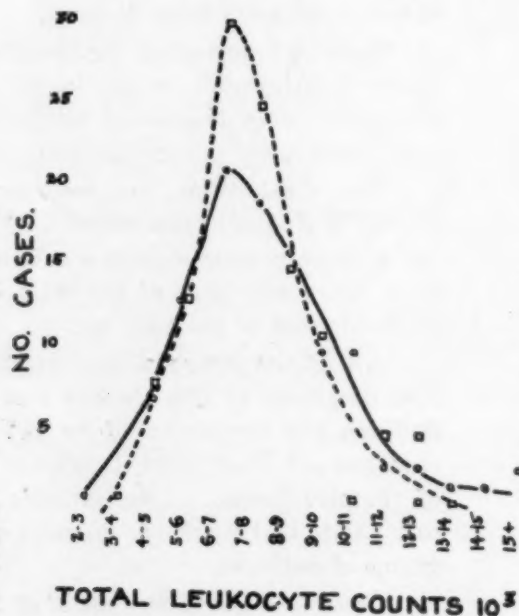


Fig. 2.

Fig. 2.—A comparison of the total leucocyte counts in 110 cases of fusospirochetal stomatitis (solid line) with 110 cases of periodontal disease (dotted line).

The total leucocyte counts of 110 patients with various types of periodontal disease were compared with the total leucocyte counts of patients with fusospirochetal stomatitis. These data are depicted in Fig. 2.

The range of the counts in both groups was similar. Although more of the total leucocyte counts in patients with periodontal disease were between 5,000 to 8,000 cells per cubic millimeter, the general distribution of the total leucocyte counts in both groups of cases was similar.

The percentage contribution of the lymphocytes and the neutrophils to the total leucocyte count is shown in Fig. 3. Except for an occasional count, the percentage of lymphocytes and neutrophils fell within the generally accepted range of values. A comparison of these percentage values with those obtained from the periodontal cases did not suggest that a relative lymphocytosis was a prominent feature of fusospirochetal stomatitis.

Since the absolute cell counts afford an appreciation of slight hematologic changes which are not so readily appreciated in the percentage values, the absolute neutrophile and lymphocyte counts for the 110 cases of fusospirochetal disease were determined. These data are shown in Fig. 4.

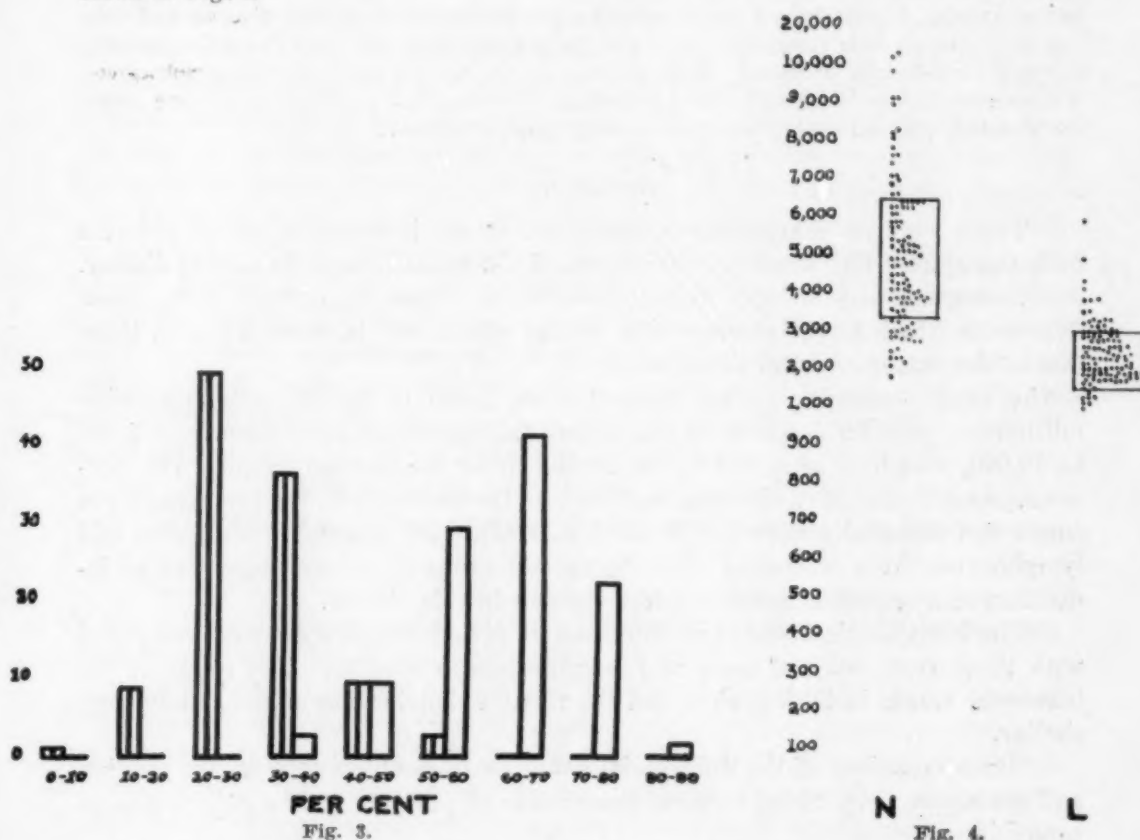


Fig. 3.

Fig. 3.—Percentage distribution of the neutrophils (unhatched columns) and lymphocytes (hatched columns) in the differential blood counts in 110 cases of fusospirochetal stomatitis.

Fig. 4.—Distribution of the absolute neutrophile (N) and lymphocyte (L) counts in 110 cases of fusospirochetal stomatitis. The rectangular areas surround absolute cell counts which were within the generally accepted range of normal values.

The absolute neutrophile counts varied from 13,345 to 1,885, and the absolute lymphocyte counts from 5,871 to 1,040 cells per cubic millimeter. According to Kolmer, the normal range for the absolute neutrophile cell count is from 6,500 to 3,150 and the normal range for the absolute lymphocyte count is from 3,000 to 1,500 cells per cubic millimeter. The counts falling outside of the rectangular areas in Fig. 4 represent absolute neutrophile or lymphocyte cell counts outside the normal range of values.

The absolute neutrophile and lymphocyte counts for the 110 periodontia cases were analyzed to determine the number which were above or below the generally accepted range of normal values which were previously given. These findings are compared with those from the patients with fusospirochetal stomatitis in Table I.

TABLE I. FREQUENCY OF ABNORMAL ABSOLUTE NEUTROPHILE AND LYMPHOCYTE COUNTS IN FUSOSPIROCHETAL STOMATITIS AND PERIODONTAL DISEASE

| CASES | NEUTROPHILES | | LYMPHOCYTES | |
|-------------------------------|------------------------------------|------------------------------------|------------------------------------|------------------------------------|
| | ABOVE 6,500 CELLS PER CU.MM. | BELOW 3,150 CELLS PER CU.MM. | ABOVE 3,000 CELLS PER CU.MM. | BELOW 1,500 CELLS PER CU.MM. |
| | | | | |
| Fusospirochetal disease (110) | 19 | 23 | 22 | 8 |
| Periodontal disease (110) | 9 | 17 | 21 | 4 |

Forty-two of the absolute neutrophile counts of the patients with fusospirochetal infection were outside the generally accepted normal range as compared to 26 in the periodontal disease group. The higher-than-normal and the lower-than-normal counts were about equally distributed in the patients with fusospirochetal disease while there was a 2:1 ratio between the low to high absolute cell counts in the periodontal disease group. There was no suggestion of any significant change in the absolute neutrophile count.

Thirty of the absolute lymphocyte counts in the patients with fusospirochetal stomatitis, as compared to 25 in the periodontal group, were not in the generally accepted range of normal values. Twenty-two of the 30 absolute lymphocyte counts in fusospirochetal disease, and 21 of the absolute lymphocyte counts in the periodontal groups were above the generally accepted normal range of values. These findings do not indicate that a relative lymphocytosis is a common feature of fusospirochetal stomatitis; however, when abnormal lymphocyte counts are obtained, they are usually above the normal range of values.

SUMMARY

There were no characteristic alterations in the hemograms of 110 patients with fusospirochetal stomatitis which would aid in the diagnosis of this disease. The hemogram may be very useful, however, in ruling out certain of the blood dyscrasias which might produce oral lesions which may be confused with those due to the fusospirochetal organisms.

The total leucocyte counts ranged from 2,900 to 16,000 cells per cubic millimeter, with 78 per cent of the counts falling within the range from 5,000 to 10,000, which is accepted by Wintrobe to be the normal range. The percentage contribution of the neutrophiles and lymphocytes to the total leucocyte count was essentially normal. When the absolute cell counts, neutrophiles and lymphocytes, were compared with the normal range of values, there was no indication of a consistent relative lymphocytosis in this disease.

The hematologic findings of 110 cases of periodontal disease were compared with those from the 110 cases of fusospirochetal stomatitis. The range of the leucocyte counts in both groups and the relative distribution of the counts were similar.

The comparison of the differential count findings obtained from the gingiva and the finger prick blood in these two groups of patients will be reported separately.

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MANDIBULAR LESION AS FIRST EVIDENCE OF MULTIPLE MYELOMA

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D. ALAN SAMPSON, M.D.‡

MYELOMA is a fairly rare tumor of bone, occurring in persons over 40 years of age. Its occurrence is twice as frequent among males as among females. Race is not significant. The tumor arises from the bone marrow, metastasizing to other bones. Flat bones containing red marrow are first involved. Solitary lesions rarely occur; over 90 per cent of cases present multiple tumors. Pain is an outstanding symptom. It is intermittent, insidious, and radiating at the onset, passing off only to recur and progressively increase until the fatal termination of the disease.^{1, 2} Roentgenograms show multiple punched-out radiolucent areas, especially in flat bones containing red marrow. Of these bones, the calvarium, ribs, vertebrae, sternum, clavicle, and pelvis may be involved. Lesions in the long bones appear late in the course of the disease. The mandible is rarely the site of the primary lesion. The case reported by Wolff and Nolan⁷ is the only one we have found in the literature. The trabecular and cortical bone is resorbed. The periosteum is perforated occasionally with the growth invading the surrounding soft tissues. Pathologic fractures occur in 62 per cent of cases.^{2, 3} Bence-Jones protein appears in the urine of 75 per cent of cases with multiple myeloma. The presence of this protein in the urine is diagnostic of multiple myeloma. Bence-Jones protein may also be demonstrated in the blood.

Destruction of the bone marrow may lead to a progressive myelophthisic anemia. The tumors are elastic, giving a parchmentlike crepitus on pressure not unlike what is felt when a cyst is palpated.

Multiple myeloma may be confused with lumbago, spondylitis deformans, Pott's disease, osteomalacia, osteitis fibrosa cystica, cystic tumors of the jaw, and eosinophilic granuloma.⁶ The disease terminates fatally, with an average duration ranging from one to two years. Roentgen therapy may temporarily retard the growth of lesions which may melt away with recalcification, but, in spite of treatment, new lesions appear elsewhere.^{1-3, 5}

A 57-year-old white married woman consulted her dentist early in December, 1943, for relief of pain in the right mandibular molar area. Local examination revealed loosening and caries of the first and second molars. The dentist removed both teeth. The pain subsided and the patient had no further discomfort for about three weeks, when she noticed a slight swelling on the right side of the mandible. The pain and swelling increased. She again sought the advice of her dentist in February, 1944. Examination revealed enlargement of the right lateral surface and inferior border of the mandible from the angle to the second premolar area. The dentist, suspecting bone disease, referred her to the Oral and Dental Section of the Surgical Service of the Episcopal Hospital, Philadelphia, for treatment.

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When admitted to the hospital, March 10, 1944, the patient was well nourished and on physical examination exhibited no pertinent findings other than the mandibular tumor. Her past medical history was irrelevant. Fever was absent. The pulse and respiration were normal. Blood study revealed 4,460,000 erythrocytes; 13.5 Gm. hemoglobin per 100 c.c. of blood; 9,100 leucocytes, 14 per cent lymphocytes, 1 per cent monocytes, 1 per cent eosinophiles, 84 per cent polymorphonuclear neutrophils, 52 per cent segmented, 32 per cent nonsegmented. The bleeding time was $1\frac{1}{2}$ minutes, and the clotting time $4\frac{1}{2}$ minutes. The sedimentation rate was 28 mm. for the first hour. The blood Kahn test was negative. The urine was negative for Bence-Jones protein.

Roentgen examination of the mandible revealed a bone defect 5 cm. in length on the right side, involving the entire thickness of the body and anterior one-third of the ramus except for a margin of bone along the alveolar border approximately 4 mm. in thickness. This thin shell of bone was fractured in two places. There was no evidence of new bone formation. The roentgen appearance was suggestive of tumor formation with pathologic fracture (Fig. 1). The tumor mass in the right mandible was excised March 11, 1944. At the same operation two holes were drilled in each major fragment and by means of vitallium pins inserted into them, a previously prepared acrylic splint was applied externally, of the type reported by Meloy and Gunter.⁴



Fig. 1.—Osteolytic lesion in mandible before operation, Feb. 16, 1944.

The pathologic report by Dr. W. P. Belk, Pathologist to the Episcopal Hospital, follows: "The specimen measures about 4 by 3 by 2 cm. It is firm with a seminecrotic center. Sections show new growth of closely packed cells, which are somewhat large with a fair amount of pink cytoplasm and dark staining excentric nuclei. The nuclei have a cart-wheel structure with chromatin condensation about the rim. The appearance is typical of plasma cells. No bone is being laid down by the tumor and only a trace of the original bone appears. The tumor is seen to extend into the surrounding soft tissues. The diagnosis is myeloma of the plasma-cell type." (Fig. 2.)

A skeletal survey was made March 16, 1944. Roentgenograms of the skull showed numerous punched-out areas in the clavarium, ranging up to 2.5 cm. in diameter. (Fig. 3.) The spine and pelvis were normal. The upper and lower extremities (not including hands and feet) were normal. The left first, sixth, ninth, and right third ribs showed punched-out

lesions. The mandible showed a radiolucent area about 1 cm. in diameter in the left premolar region (the opposite side from the fracture).

Laboratory studies at this time revealed: the urine negative for Bence-Jones protein; erythrocytes, 3,700,000; hemoglobin, 74 per cent; leucocytes, 8,300, 28 per cent lymphocytes, 2 per cent monocytes, 1 per cent eosinophiles, 69 per cent polymorphonuclears, 53 per cent segmented, and 16 per cent nonsegmented. The blood sedimentation rate was 47 mm. for the first hour.

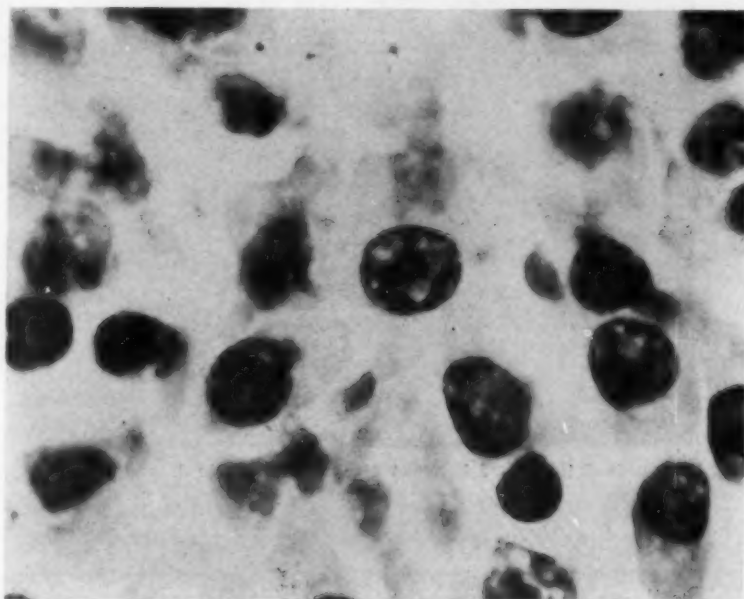


Fig. 2.—Microscopic resolution of tumor section from the mandible showing plasma cells ($\times 2,000$). (Photomicrograph, courtesy of W. M. Hammond, Research Associate in Microscopy, School of Dentistry of the University of Pennsylvania.)



Fig. 3.—Typical punched-out lesions in skull, March 16, 1944.

The patient was discharged from the hospital in good condition, March 25. Two days later, as an outpatient, she began to receive roentgen treatments to the site of the pathologic fracture of the right side of the jaw. A total of 1350 r. was given, using 200 KVP and 0.5 mm. copper filtration.



Fig. 4.—Solidly united bridge of bone at site of pathologic fracture, Jan. 5, 1945.

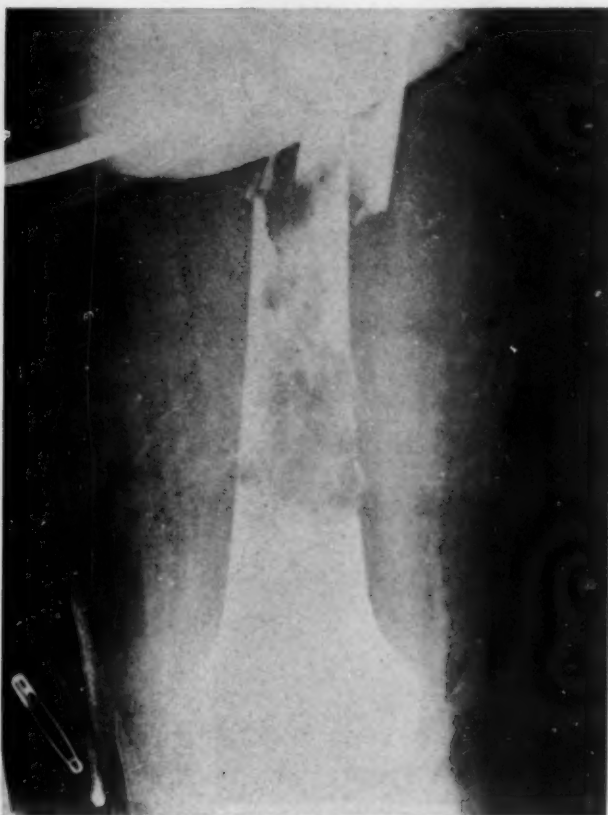


Fig. 5.

Fig. 5.—Involvement of left femur with pathologic fracture, Jan. 9, 1945.



Fig. 6

Fig. 6.—Pathologic fracture of right humerus, March 8, 1945.

Six weeks later, there was already evidence of bone regeneration and, by the end of June, union appeared to be fairly strong along the thin bridge of bone representing the alveolus, although the large defect remained. The splint, however, was not removed until October 11; it had not been taken off earlier because of the danger of refracture and because it was not until this date that the bone bridge appeared sufficiently strong. (See Fig. 4.)

Roentgen therapy was also administered to the thorax and to the skull. Under its influence there was considerable regression of the lesions. Pathologic fractures which had developed in some of the ribs united. However, when the patient again entered the hospital, Oct. 28, 1944, complaining of severe chest pain, roentgen examination disclosed pathologic fractures in other ribs. Following sedation and irradiation, she was discharged, November 18, improved.

The final readmission occurred on Jan. 2, 1945, because of pain in the left thigh and general weakness. X-ray studies showed extensive involvement of the left femur, and fracture occurred five days later when the patient moved in bed. (Fig. 5.) Bence-Jones protein was found in the urine at this time; the anemia had increased (erythrocytes 3,260,000), and the white cell count was 7,400 with 90 per cent polymorphonuclears. Fracture of the right humerus occurred on March 8, 1945, when the patient raised a pitcher of water. (Fig. 6.) Fracture of the left humerus occurred on March 26. Roentgen therapy for palliation gave relief from pain, but the patient steadily failed and succumbed on May 4, 1945.

SUMMARY

1. A case of multiple myeloma, plasma-cell type, is reported, with the largest lesion and the first to produce symptoms, located in the mandible.
2. Dentists may be the first to discover the lesions of multiple myeloma.
3. The lesion of the right side of the mandible was removed in mass and the pathologic fracture controlled by extraoral fixation and roentgen therapy. enucleation of the tumor mass, followed by roentgen treatment, allowed sufficient osteogenesis to permit the removal of the splint and to provide a comfortable, functioning jaw for the remainder of the patient's life.
4. Bence-Jones protein did not appear in the urine until after the appearance of the lesions in the long bones, approximately four months before death.

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TREATMENT OF FUSOSPIROCHETAL AND STREPTOCOCCAL INFECTIONS BY TOPICAL APPLICATION OF PENICILLIN

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PENICILLIN, a product of *Penicillium notatum*, has been extensively and successfully employed in the treatment of formerly refractory infections. Unlike most bactericides, this substance is not affected by pus, serum, or products of tissue disintegration; nor is it toxic to leucocytes. However, it has been reported to be ineffective against certain microorganisms including the gram-negative bacilli.¹ It has been assumed that long contact between drug and bacteria is necessary to obtain the therapeutic action which was considered to be bacteriostatic until recent investigations established bactericidal properties.² Since reports have been made of successful treatment of syphilis by injection of penicillin,³ and since favorable clinical observations have been made of the treatment of Vincent's infection by topical application,⁴⁻⁶ it was felt that a more detailed study might be of value.

The study, of which this is a preliminary report, is divided into: (1) In vitro study; (2) study in laboratory animals; (3) clinical observations. Solutions were prepared by dissolving 100,000 Oxford units of penicillin sodium in 10 c.c. of physiologic saline at room temperature. They were used immediately.

IN VITRO STUDY

The solution was subjected to the motility test. (Fig. 1.) This method, which has been described and evaluated elsewhere^{7, 8} uses loss of motility of the oral flora and growth of the nonmotile flora as a basis of comparison. Equal quantities of the drug and saliva from a case of acute Vincent's infection are employed. An efficient drug stops all motility at the instant of contact and inhibits growth in cultures taken from a mixture of equal parts of saliva and drug in contact for fifteen minutes.

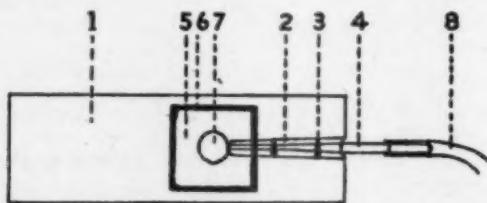


Fig. 1.—1, Glass slide. 2, Groove ground in slide. 3, Sticky wax. 4, Glass tube containing drug. 5, Cover glass. 6, Petrolatum. 7, Specimen of saliva and bacteria. 8, Rubber tubing. The apparatus is mounted on a dark-field microscope.

Penicillin sodium did not stop all motility on contact. While many spirochetes, vibrios, and motile bacilli ceased moving immediately and others showed only a sluggish and modified motion, many others continued unchanged for more than fifteen minutes.

Equal parts of saliva and the penicillin solution were placed in a test tube and allowed to stand for fifteen minutes with occasional agitation. Two loopfuls of the mixture were then transferred to a tube of hormone broth and to blood agar slants. One of the latter was incubated under anaerobic conditions. All tubes were sterile at 72 hours. Lest the findings under the microscope and in the culture tube seem incompatible, one should remember that not all of the oral flora can be grown under these conditions.

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ANIMAL STUDIES

Saliva from a case of acute Vincent's infection was centrifuged to eliminate solids. Two mature guinea pigs were given three inoculations each. In the right inguinal region, 0.1 c.c. of the saliva was injected. On the left side was injected 0.1 c.c. of a mixture of equal parts of centrifuged saliva and penicillin solution which had been prepared fifteen minutes previously. Near this site, 0.1 c.c. of a sterile physiologic saline solution was injected. A foul-smelling, gas-producing abscess developed on the right side of both animals. There was little more reaction from the saliva-penicillin in mixture than from the saline. No acute infection resulted.

CLINICAL OBSERVATIONS

Sixteen cases were observed: 14 of acute ulcerous Vincent's infection, and 2 of streptococcal stomatitis. Diagnosis was made from the subjective symptoms, clinical appearance, and microscopic examination of material from the gingivae.

All patients with Vincent's infection complained of pain, tenderness of the gingivae, fetor oris, bleeding, salivation, adenopathy, malaise, and eleven had slight elevation of temperature. The gingivae of all cases exhibited two or more areas of necrosis. The interdental papillae were eroded with necrotic edges. Several had contact ulcers of the mucosa of the lip or cheek. The gingival tissues were dark and edematous. The streptococcal infections presented a somewhat similar appearance except that the tissues were a bright red.

Bright- and dark-field smears were taken. All showed the etiologic organisms in overwhelming numbers and, in the cases of Vincent's infection, actively motile.

The patients were given 5 ounces of tap water as a rinse to remove debris. Rinsing was limited to one minute. The saliva was then excluded by cotton rolls and a saliva ejector, and the tissues were gently dried by blasts of warm compressed air. The solution was applied to the gingival crevice with a glass syringe and blunt, curved, platinum needle. Care was used to insure that the solution reached the bottom of all pockets, covered every lesion, and moistened the entire gingivae. The tissues were kept moist with the penicillin solution for fifteen minutes, when the patient was dismissed for twenty-four hours. The average quantity of solution used was 3 c.c. per patient.

No other therapeutic measures were employed. There was no removal of calculus, no mouthwashes for home use, no correction of diet, no prohibition of alcohol or tobacco, no word about toothbrushing. If the patient brushed the teeth correctly or incorrectly or not at all, he was not disturbed in his practice.

At the next visit, twenty-four hours later (four cases were 48 hours later), there was great amelioration of the symptoms in every instance. Pain and bleeding had stopped. The tissues could be squeezed without acute discomfort, and solid food could be chewed easily. Malaise, pyrexia, fetor, and ulcerations had disappeared. The temperature was normal in every case. Swelling of the lymph nodes was still present in the majority of cases. The tissues had assumed a light color, although the gingival margins and interdental papillae remained red and edematous. Smears showed a marked diminution in the number of spirochetes, averaging 1 in 5 fields in bright-field examination. No motile spirochetes were found in any of the slides examined in the dark field. In the streptococcal infections, the oral flora at this visit was similar to the others—varied, but more numerous than in a healthy mouth.

At this time, periodontal treatment was begun, consisting of prophylaxis, diet correction, toothbrush drill, equilibration of occlusion subgingival curettage, etc.

DISCUSSION

The investigation did not seek to determine how long penicillin would inhibit the microorganisms or prevent a recurrence. The function of medication is the control of the bacterial invaders. Reinfection is prevented by removing the predisposing factors, which is purely a periodontal problem. Penicillin sodium in physiologic saline solution 10,000 units per cubic centimeter functioned well in these cases.

In this concentration, there is no evidence of toxicity or destruction of the hard and soft tissues, nor any action on restorations. The presence of organic

and inorganic debris seemed to interfere with its efficiency in vitro, but not in vivo. The speed of action, power of penetration, freedom from undue specificity, efficiency at mouth temperature, and solubility in water were eminently satisfactory. The taste is not particularly objectionable. There was no staining.

Penicillin has two disadvantages. It is unstable and can be stored for a limited time only in a refrigerator. In solution, its life is extremely limited. The cost is high in comparison to that of other medicaments used in the treatment of these infections.

The results of the experiments in vitro would indicate some actual bactericidal properties of this substance. Perhaps the difference in action on the motile and on the nonmotile oral flora might provide a means for investigating the role of each in the etiology of Vincent's infection.

CONCLUSION

While this investigation has been too limited for definite conclusions to be drawn, it would seem that penicillin solutions have value in the treatment of acute streptococcal and fusospirochetal infections of the mouth by topical application. There is no evidence, however, that it is superior to a number of other drugs on which we have previously reported.

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UNILATERAL LACRIMATION ASSOCIATED WITH CHEWING

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THE phenomenon of unilateral lacrimation associated with chewing has been described under the syndrome of "crocodile tears." Essentially, the condition consists of a paroxysmal lacrimation every time the patient salivates during eating. It was called the syndrome of "crocodile tears" by Bogorad¹ because the crocodile was formerly believed to weep hypocritical tears while devouring its victim. It occurs only after peripheral facial nerve palsies, or injuries to this nerve, e.g. accidental severance. It is not to be confused with lacrimation after Bell's palsies in which there is a paralysis of the eye muscles or an ectropion permitting tears to run out of the conjunctival sac.

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Ford² reported four cases of lacrimation associated with chewing and observed that the tearing was associated with salivary flow. The lacrimation started only after the salivary glands were stimulated, such as by taste and smell, and were not influenced by any other circumstances. Russin³ reported two cases in which lacrimation occurred every time the patients ate any salty, acid, or bitter foods. Masticatory movements had no effect on the lacrimation nor did aromatic spirits of ammonia and other strong odors.

The following case of lacrimation is presented because of the rarity of this condition.

Dr. Philip Adalman, an ophthalmologist of New York City, invited me to see a 72-year-old man who complained of redness of the conjunctiva, lacrimation, and pain in the left eye and forehead when he chewed his food.

His illness began about twenty years before when he was walking in the street on a day in which the humidity was high; he felt his nose "clog up." He put his finger in his left nostril and experienced a sharp "dancing" pain on the left half of his face, in the eye and forehead. There was no pain in the nose, gums, or tongue. The pain reappeared two or three times in the following two weeks. This was ameliorated by rubbing alcohol on his face. He had had no trouble chewing his food with dentures twenty years ago. He was certain that the discomfort came after he had worn the teeth for about one year. He discovered he could avoid pain by chewing his food on the right side. As time progressed, the pain became more intense and during the past six or seven years it had become very severe.

There was no history of injury to the head or trauma in extraction of the teeth. For many years, he had become progressively deaf. Five years after the onset of the facial pain, he was stricken with an obscure condition with symptoms of drowsiness, retention of urine, weakness in the extremities, and dizziness. The rest of the medical history was negative.

Examination disclosed a well-nourished man with a somewhat enlarged heart. The blood pressure was 170/100 and there was evidence of arteriosclerosis. He had neurologic signs indicating disease of the nervous system. The left eyeball was more prominent than the right, and the left pupil was larger than the right. The left corneal reflex was absent. There was diminished sensation for pain, temperature, and touch in all three divisions of the left trigeminus. There was redness of the conjunctiva and lacrimation in the left eye and pain in the area of distribution of the first division of the left trigeminus under the following conditions: vigorous chewing movements, such as eating; pressure on the anterior palatine pad by rubbing the finger over the area; rubbing the left nostril or blowing the nose; tasting a hot pepper; electrical stimulation of the left forehead; deep pressure of the left cheek sufficient to cause pain; and attempt to read small print.

The blood Wassermann was negative and urinalysis was normal.

The dental examination revealed nothing abnormal. The dentures were in good occlusion and the physiologic rest position was within normal limits. He had had two sets of dentures in the last twenty years. The second was made because the original dentures were loose. The mucous membrane was normal in color and the anterior palatine pad area showed no signs of abrasion or erosion.

The congestion, lacrimation, and facial pain could not be elicited under the following conditions: pressure on the right and left posterior palatine area, stimulating the muscles of mastication by moving the jaw up and down or side to side without the dentures; pressure on the mental foramen, biting on right or left side only with dentures in place.

COMMENT

From a dental point of view, it was noteworthy that only stimulation of the anterior palatine pad area brought about congestion, lacrimation, and pain. The effects were best produced by rubbing with a finger, or denture pressure through food mastication. While the above symptoms could be produced by reading small print and blowing the nose, the dental complaint was the more important because the chewing apparatus is needed for daily sustenance.

The pad area was definitely localized as the trigger point by the fact that unilateral biting and movements of the muscles of mastication did not reproduce the foregoing syndrome. When there is independent biting either on right or

left side, there is no compression or rubbing on the pad area. With normal chewing the force of mastication produces a pressure because of the closeness of adaptation of the dentures on the yielding mucosal tissues. This phenomenon is of special interest to the prosthodontist because it exhibits objective signs and subjective symptoms when there is impingement of certain nerve areas in the mouth.

This case differs from those reported in the literature in that, associated with lacrimation, there was also congestion and pain. Whether this can be attributed to a stimulation of both the autonomic and sensory systems is difficult to say. This interesting condition may also be associated with a disturbance in the left trigeminus, i.e., either a trigeminal neuritis or a neuralgia.

There was no doubt that this patient had a disease of the nervous system with the left fifth cranial nerve involved. In one attack there may have been a neuritis of the left trigeminus and probably many of the nerve fibers were degenerated. During the process of repair the regenerated fibers branched and grew in all directions, losing their original anatomic arrangement.⁴ It can be easily understood that some of the sensory fibers may have regenerated and grew into the sheath containing autonomic fibers. Under such conditions an impulse meant for one group of structures would spread by diffusion and axon reflexes to other structures. The result would be diffuse effect as in this case. Whenever this patient began to eat and pressure was exerted, impulses spread to the ophthalmic division, causing formation of tears, congestion, and sensations of pain. This is analogous to crocodile-tear syndrome due to a lesion of the facial nerve involving the geniculate ganglion,² the auriculotemporal syndrome,⁵ the synkinesias following Bell's Palsy,⁶ and the pseudo Graefe phenomenon.⁷

The other possibility is that this patient had a trigeminal neuralgia. In this condition, when the ophthalmic division is involved, there is lacrimation and congestion of the conjunctiva as seen in this case. There are trigger points, which this patient seemed to have over the nostril, gums, forehead, and face. The chief objection to trigeminal neuralgia, however, is that there is definite evidence of disease of the trigeminal nerve, that is, sensory changes in the face and loss of corneal reflex. These signs indicate an involvement of the trigeminal nerve and therefore take it out of the category of a neuralgia.⁸

SUMMARY

A case of unilateral lacrimation, congestion, and pain associated with chewing is described. It differs from the ones reported in the literature in that there is an associated congestion and pain.

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STOMATITIS DUE TO CHEWING LEAVES OF POISON IVY

REPORT OF A CASE

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THE dermatologic manifestations of poison ivy are commonly seen during the summer months. Oral manifestations of poison ivy are, however, rare. A search of the dental literature revealed nothing about stomatitis venenata due to poison ivy. The rarity of the oral manifestations due to sensitivity to the *Rhus toxicodendron* may be due to the fact that the leaves of this plant are seldom eaten, particularly by those who are sensitive to poison ivy, and because the mucous membranes are less sensitive to the oleoresins of the poison ivy plant.¹

The following case of *Rhus toxicodendron* stomatitis is reported because of the rarity of this form of stomatitis venenata and the diagnostic difficulties such a case presented.

CASE REPORT

Miss L. F., a 19-year-old white woman who had had severe attacks of poison ivy dermatitis in the past, ate three leaves of poison ivy on the advice of friends who told her that this would prevent further attacks. Several of her friends also ate some of the leaves, which they had done before with no injurious effects.

Two days later the patient developed swollen lips, a sore throat, submaxillary adenopathy, and a temperature of 100° F. (oral). The third day her mouth became intensely painful. Examination at this time revealed edematous lips with several small vesicular lesions. The tongue was so swollen and so painful that normal speech was difficult. The gums were hypertrophied and inflamed with shallow erosions in the posterior part of the mouth. The palate was covered with debris due to superficial necrosis of this tissue and forced immobility of the tongue. There were several shallow erosions of the palate. The buccal mucosa was inflamed but devoid of other lesions. The fauces and pharynx were inflamed. Fetor ex ore was marked and the patient complained of a foul taste. There was moderate submaxillary and cervical adenopathy. The history of ingestion of any drugs, chemicals, or any other substances not usually eaten by the patient was negative. An oral smear revealed gram-positive diplococci, gram-negative cocci, and gram-negative bacilli. The white blood count was 12,500 cells per cu.mm., and the hemoglobin was 98 per cent (15.6 Gm. = 100 per cent).

The fourth day the patient developed the typical lesions of poison ivy dermatitis on her arms, hands, and legs. The onset of the skin lesions aided the patient to recall that she had eaten the three leaves of poison ivy. The diagnosis was then established. Dr. John Stokes, the Professor of Dermatology at the Hospital of the University of Pennsylvania, confirmed the diagnosis. None of the patient's friends showed any manifestations of poison ivy sensitivity in the mouth or on the skin.

Until the diagnosis had been established, the patient was given anesthetic troches before meals so she could eat. After consultation with the Dermatology Department the patient was given a mouthwash of 1:1,000 tannic acid in water every hour. After two days of this therapy the oral symptoms greatly decreased; on the third day of this treatment the oral lesions had completely disappeared. The dermatitis was treated with calamine lotion and the skin lesions cleared within a week.

*Dental Intern, Hospital of the University of Pennsylvania. Dr. Sassaman is now serving in the Navy.

DISCUSSION

There is some therapeutic basis for eating poison leaves to desensitize the patient to this plant. Gilmore² reported that this method was used by American Indians. Dakin³ reported the use of this method by the rural inhabitants of New Jersey in 1829 with good results. Later both Dakin³ and Silvers¹ studied this method of immunization. Both reported cases of pruritis and following the ingestion of poison ivy leaves. Cooper⁴ advised eating small fresh leaves in the spring as a method of prophylaxis. He reported good results following this method. McNair⁵ observed violent gastrointestinal disturbances and proctitis following oral administration of poisoning leaves.

The dermatologists⁶ at the Hospital of the University of Pennsylvania feel that the oral administration of raw leaves of poison ivy for the prophylaxis of poison ivy dermatitis is not justified in view of the possible severe oral, gastrointestinal, and anal disturbances.

SUMMARY

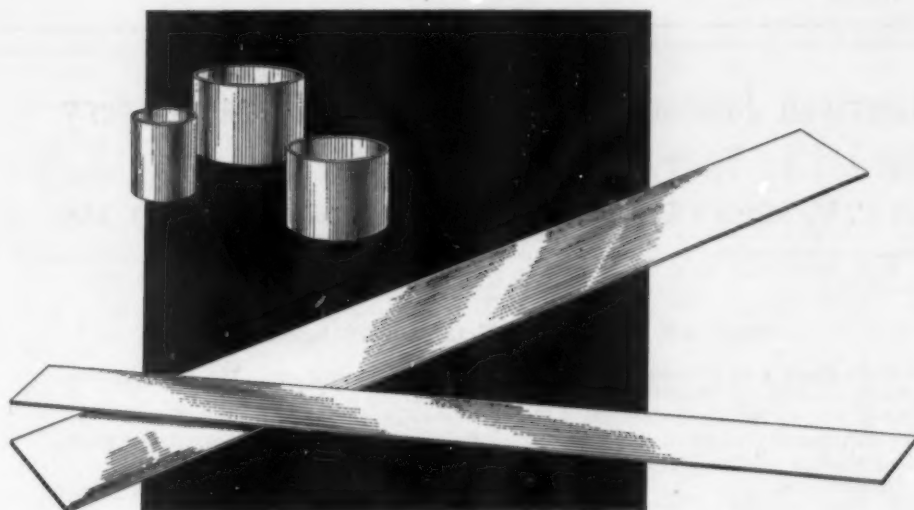
A case of stomatitis venenata due to chewing three leaves of poison ivy has been presented along with a discussion of the oral prophylaxis of poison ivy dermatitis.

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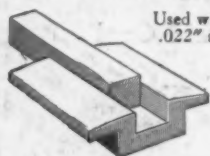
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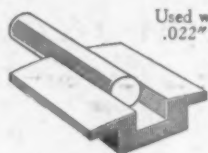
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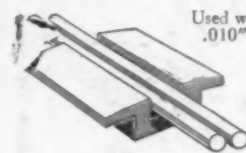
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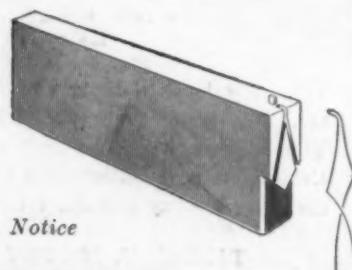
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BERT G. ANDERSON, D.D.S.,* AND GEORGE A. KENTROS, D.M.D.,†
NEW HAVEN, CONN.

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1. Three donors. Transfusions (a) about 350 c.c. to be given within thirty-six hours before operation followed by clotting time. If clotting time is reduced, transfusion (b) of at least 500 c.c. to be given within eight hours of operation followed again by clotting time. Transfusion (c) to follow operation if necessary and donor (a) to be held in reserve for second donation if necessary. The presence of slight allergic reaction or serum sickness to be no contraindication.

2. Fresh muscle, human preferably, to be sewed into the wounds to aid hemostasis.

3. Injections of theelin, thromboplastin, etc., to be withheld unless definite postoperative emergency arises.

The patient received a total of 1,100 c.c. of blood before the extraction of the teeth. Ten teeth were removed and care was exercised in order to produce a minimum amount of trauma. Following extractions, straight incisions were drawn through to the bone, both buccally and palatally to the open sockets. The septal gingivae were then removed, as well as a sufficient amount of alveolar process to permit the smooth edges of the palatal and buccal gingivae to be closely approximated and held together with continuous lock sutures. The maxillary gingival edges were also brought into apposition and closed with a continuous lock suture without the application of muscle tissue in order to serve as a control.

Laboratory Data.—At the time of admission, laboratory blood examination showed the following conditions: red blood cells, 3,490,000; hemoglobin, 13 Gm.; white blood cells, 6,200; clotting time, venous blood, 20 minutes. Following transfusion of 1,100 c.c. of whole blood, the laboratory blood examination showed: red blood cells, 4,490,000; hemoglobin, 15 Gm.; clotting time, 11 minutes.

Patient had no significant bleeding immediately following the operation, but several hours later it was observed that the maxillary right molar socket and both groups of mandibular sockets began to bleed fairly rapidly. Impression compound saddles were used to obtain pressure over the alveoli and to hold thromboplastin packs in place. Bleeding became intermittent. After two days the muscle tissue showed evidence of sloughing and necrotizing, and a secondary hemorrhage followed, which was more difficult to control than that of the

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mandibular alveoli in which no muscle tissue had been placed. This blood loss necessitated the use of supportive transfusions of whole citrated blood to offset the possibility of circulatory collapse and shock. It was necessary, however, to place a great deal of reliance for checking this bleeding on the pressure obtained with the aid of tightly fitted saddles. The patient was also put on therapy for anemia due to acute blood loss.

The oozing of blood from the sockets was arrested after nine days. On the eleventh day, sutures and packs were removed and the patient was discharged from the hospital fourteen days following the operation.

CASE 2.—A 13-year-old white boy, D. S., was brought to the emergency room of the New Haven Hospital on April 22, 1945. The patient was a known hemophiliac who had been admitted for many episodes of bleeding since birth, the last admission having been two and one-half years previously. The hemorrhages in the past had been intracranial, intra-articular, and retroperitoneal. Since his previous admission, the patient had been in fairly good health except for occasional mild traumatic ecchymoses and hemarthroses.

According to the mother, on the day before admission, the boy had been taken to an exodontist for the extraction of three six-year molars. The school dentist had suggested the immediate need for extraction of the maxillary left first molar, and the two mandibular molars. The exodontist was not told that the child had a history of hemophilia and was known to be a "bleeder." The mother realized that the boy might not stop bleeding easily following the extractions but she was influenced by the fact that on several occasions deciduous teeth were removed without significant postoperative bleeding. The boy had been hospitalized for these earlier extractions and had been given prophylactic transfusions prior to these operations. She hesitated to inform the exodontist of her child's blood condition, fearing that he might insist upon hospitalization which would involve the unnecessary use of a hospital bed.

The surgery was accomplished under nitrous oxide anesthesia. When the patient left the office, no significant bleeding was observed. About five hours later, however, a continuous oozing of blood in the oral cavity was noticed, followed by nausea and vomiting of a coffee-ground material. The following day the bleeding still continued and the boy was suffering from extreme thirst, dizziness, weakness, and pallor. When brought into the emergency room, the patient had gone into syncope.

Treatment of Shock.—The immediate measures taken were those of combating shock. Patient was immediately put in the Trendelenburg position and intravenous therapy was instituted. Infusions of saline, pooled plasma, and whole blood were given. At the time of admission to the emergency room, examination revealed the following: temperature, 100.4° F.; pulse, 160; respirations, 50; blood pressure, 85/40. The red blood count was 1,390,000, hemoglobin 4.2 Gm., and white blood count 10,900. The skin was very pale and waxy, the extremities, cold and moist. The lips were a very light pink. Oral examination showed ischemic gingivae, actually being without color. The mouth was filled with large, stringy, adhesive clots, making it difficult to designate the sites of extraction.

After the transfusions and intravenous therapy, the following laboratory findings were obtained; temperature, 101.3° F.; pulse, 124; respirations, 30; blood pressure, 120/70; and coagulation time, 29 minutes. The red count was now 3,730,000, hemoglobin 8.5 Gm., and white count unchanged. Now that there was an improvement in the systemic condition of the patient, control of bleeding from the sockets was attempted.

Local Treatment (Fibrin Foam and Thrombin).—All the loose, clotted matter was removed from the mouth and considerable bleeding could now be seen from both lower first molar sockets and a lesser amount from the upper left first molar socket. A supply of fibrin foam and thrombin was obtained for the purpose of arresting the hemorrhage, after packing of the sockets with sterile gauze and application of pressure for forty-five minutes had failed. The fibrin foam was cut into the desired sizes and was saturated with a solution of saline and thrombin. This was then applied into the alveoli of the lower molar sockets. The upper socket was left as a control, inasmuch as a fairly adequate clot seemed to have formed in it. The material was maintained in position by placing small square sponges over the area and having the patient bite down on them.

The following day it was necessary to control bleeding from the upper left molar socket. Examination showed that considerable oozing was taking place from the mesial and distal aspects of the inadequate clot. Again a fresh supply of fibrin foam saturated with thrombin and saline was placed over the socket and a portion was tamped into the deficient mesial and distal portions of the clot. The patient was instructed to bite down on small, sterile sponges.

Hemorrhage was arrested temporarily in the upper socket but two days later a persistent ooze became apparent. This time the fibrin foam was held in position with the aid of a saddle of impression compound. When the compound was chilled and trimmed, it included the teeth on either side of the sockets of the left side. A final wash was taken extending to the mucobuccal fold of the upper teeth, and a snug fit at the periphery was obtained. The negative impression of the lower molar socket was relieved so that it would not traumatize the already formed clot. The fibrin foam pack was inserted over the upper molar socket and the saddle was inserted. The patient now had a more closed position of the jaws and did not complain of temporomandibular pain as he had before, due to the opened bite caused by the gauze packs.

The saddle was removed after twenty-four hours and a complete arrest of hemorrhage was seen. The clot appeared adequate and extended slightly out of the alveolus. The patient was discharged in good condition and free from bleeding on the tenth day of his hospitalization and was asked to report to the dental clinic for observation in a few days.

Comparison of Fibrin Foam and Thrombin With Human Muscle as Hemostatics.—The use of muscle as a hemostatic agent was introduced to neurosurgery by Cushing. Ingraham and Bailey have found fibrin foam with thrombin to be superior in several respects to muscle for this purpose. They state:

"The tissue reaction to fibrin foam with thrombin is much less than that elicited by muscle. Furthermore, the surgeon who uses muscle must content himself with the limited supply which he may obtain from the temporal muscle or perform another procedure to obtain additional material from the gastrocnemius or other muscle, or depend on the chance that muscle may be secured from a concomitant operation. On the other hand, fibrin foam with thrombin is ready in any amount and can be placed on the instrument table with as little concern as is given to the provision of an adequate supply of instruments."

Conclusions.—Although muscle may be an effective hemostatic elsewhere in the body, its use in a septic field such as the oral cavity may be contraindicated. In this instance, the muscle underwent rapid putrefaction, which necessitated a reopening and retrimming of the edges for resuturing of the wound. These results indicate that fibrin foam and thrombin were more effective than muscle in controlling the hemorrhage. From the comparison of these two cases, it appears that the further use of fibrin foam and thrombin is indicated for the arrest of severe hemorrhage in the oral cavity.

The fibrin foam and thrombin used in this work has been prepared under contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Harvard University.

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EXPERIMENTAL CHRONIC FLUORINE INTOXICATION: EFFECT ON BONES AND TEETH

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MUCH experimental and clinical work has been done on the effect of chronic fluorine intoxication on teeth, but little attention has been paid to the changes in the bones. The work herein presented has been particularly stimulated by the reports of Moeller, Flemming, and Gudjonsson¹ and the excellent monograph of Roholm.² While Moeller, Flemming, and Gudjonsson conducted the clinical and roentgenologic studies on workers of a cryolite factory in Denmark, Roholm described the chemical and histologic changes of the bones of two of these workers who died of intercurrent diseases. Severe sclerotic changes of all bones, but mainly the vertebrae, pelvis, and ribs, were noted in 86.8 per cent of the workmen. The chemical and pathologic examination of the bones revealed that the bone changes resulted from a chronic fluorine intoxication. The bones were three times the normal weight, and their thickness appeared greatly increased, due to broad layers of new periosteal bone, which made the surface rough and chalky. The fluorine contents of the skeleton were about sixty times the normal amount, and the lungs contained a remarkable amount of fluorine.

The microscopic examination showed excessive periosteal and endosteal bone formation with an irregular globular calcification of the matrix. The trabeculae of the sclerotic bones that encroached upon the medullary cavity had osteoid zones of normal width. A very moderate osteoclastic resorption has been observed here and there. A widespread calcification of the ligaments was noticeable.

Almost simultaneously with Roholm, Bauer, Bishop and Wolff³ studied microscopically and chemically the bones of a 48-year-old workman in a superphosphate fertilizer factory who died of a cardiac failure. The fact that the fluorine ash value was from ten to twenty times the normal value confirmed that the osteosclerosis was due to a chronic fluorine intoxication. The microscope revealed excessive periosteal and endosteal bone deposition which led to a thickening and condensation of the bones.

In England, Wilkie⁴ has observed in roentgenograms very distinct bone changes, due to chronic fluorine intoxication, in two workmen from an aluminum fluoride factory. Tooth changes in the cases reported were not present, as these workers had been exposed as adults to the excessive amount of fluorine. Chronic fluorine intoxication, however, affects only tooth buds. The erupted teeth show then the well-known lesions of "mottled enamel."

These cases of bone involvement, due to chronic fluorine poisoning, resulted from the inhalation of gaseous fluorine compounds. However, most of the cases of epidemiologic chronic fluorine intoxication in man and animals are due to ingestion of food containing harmful amounts of fluoride compounds, or covered by it, but mainly by drinking water with a higher percentage of fluorine than normal. The chief interest of investigators has been concentrated on the study of the lesions of the teeth of children, brought about by the intake of excessive fluorine during tooth formation. No attention has been paid in this

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country, where mottled enamel is endemic in many parts, to the question whether the dental fluorosis of man occurs directly or is indirectly associated with skeletal changes, notwithstanding partially confirmative reports from foreign countries in recent years.

Shortt and co-workers,⁵ and later, Pandit⁶ in India, and Oekerse⁷ in South Africa found in districts where the fluorine content of water was ranging from 4 to 13.9 parts per million that adults with mottled enamel also revealed changes of the bones. The spine became rigid "like a continuous column of bone," exostoses thickened the bones, calcification and ossification of the ligaments and tendon insertions were common. The patients died from intercurrent diseases.

Recently, Linsman and McMurray⁸ observed a fluoride osteosclerosis and excessive mottled enamel in a white soldier, who was born and raised in an area of this country in which mottled enamel is endemic. The soldier died of a chronic bilateral pyelonephritis with acute pyonephrosis, and the chemical analysis of bones and teeth verified the diagnosis of chronic fluorine intoxication. The author considers the kidney lesion, also, as the possible result of chronic fluorine poisoning. The osteosclerosis affected an encroachment upon the bone marrow so that a secondary anemia developed.

However, no reports on coinciding involvement of teeth and bones in fluorine poisoned and growing children were available until 1942, when Kemp, Murray, and Wilson⁹ presented a study on "Spondylosis Deformans, in Relation to Fluorine and General Nutrition." These authors radiologically examined twenty-two children, from 9 to 14½ years of age, with moderate to severe dental fluorosis, in parts of England where the drinking water contains from 0.3 to 1.2 parts per million of fluorine. Unfortunately, they limited the investigations to the vertebrae of the children. They found "disturbances in the natural ossification of the spines" of all these young patients, thus revealing early signs of spondylitis deformans. The authors concluded from these findings that "fluorine in the water supply may influence the development of such defects, especially when it is associated with defective nutrition." In their summary they pointed particularly to eight children who showed severe mottled enamel and disturbances of ossification.

A coincidence of "mottled enamel" and bone lesions in children had to be expected, on account of such findings in human beings and animals, reported by French authors. Men and animals in Algiers, Tunis, and Morocco were exposed to the high fluorine content of drinking water in early age when the teeth were developing and the bones growing. The chronic fluorine intoxication affected the teeth and also the bones of the extremities, skull, and jaws.

The occurrence of tooth and bone involvement at the same time has also been proved by experiments on rats, pigs, sheep, and calves. The teeth revealed the characteristic changes of mottled enamel first described in detail by McCollum, Simmonds, Becker, and Bunting.¹⁰ However, there is no uniformity of opinion as to the nature of the bone lesions. Some authors reported a rickets-like systemic disease; others denied the presence of abundant osteoid tissue but did not hesitate to speak of a "generalized condition resembling osteomalacia." Osteoporosis and exostoses have been described in above-mentioned animals, while "osteosclerosis," a product of excessive endosteal and periosteal bone formation with a heavy granular calcification, has been observed by Sutro¹¹ in rats only. A similar "osteosclerosis" with a qualitative abnormal calcification by coarse granules has been found in rats by Roholm, together with a widespread calcification of ligaments and tendon insertions, thus resembling the bone lesions in chronic fluorine intoxication in men.

The present study attempts a clarification of the nature of the skeletal changes in chronic fluorine intoxication and their correlation to tooth lesions.

The questions I have tried to answer are as follows:

1. Does chronic fluorine intoxication affect the skeleton of dogs?
2. Does vitamin D added to the fluorine diet alter the changes of the bones and teeth?



Fig. 1.—Roentgenograms of tibiae and fibulae of dogs of the first group. A, Control dog. B, Dog 4 after 96 days peroral ingestion of about 0.1 Gm. sodium fluoride daily. Excessive sclerotic periosteal bone, osteoporosis of the spongy part.

3. Is fluorine excreted with the mother's milk and does it affect the bones of puppies?

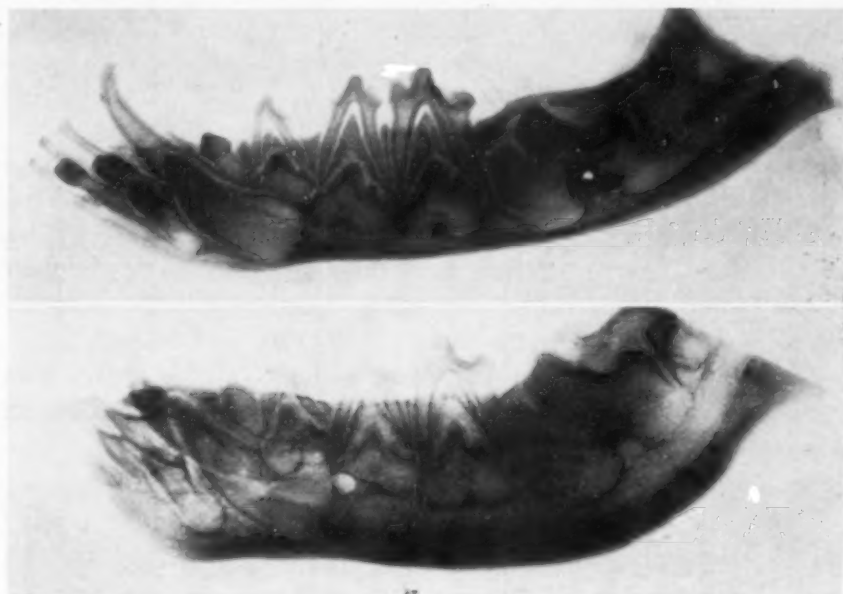
4. Do the bone changes start before, simultaneously with, or after, the tooth changes begin?

Sixteen dogs of three litters were employed in the experiments. The pertinent data are presented in Table I.

The dogs, placed in separated spacious rooms with good sunlight, were kept as much as possible in open air in the daytime. The puppies fed a fluorine diet generally reacted with symptoms of indigestion for the first four or five days, but regained their appetite and liveliness quickly. Thereafter, the experiments remained undisturbed. The dogs were killed by cutting the carotid artery under ether narcosis.

At autopsy, the endocrine glands, the kidneys, and the following bones of the experimental and control dogs were removed: the long bones, ribs, jaws with the teeth, vertebrae, scapulae, and parts of the skull. The bones were examined roentgenographically, decalcified in 5 per cent HNO_3 , embedded in celloidin, sectioned, and the sections stained with hematoxylin and eosin.

A.



B.

Fig. 2.—Roentgenograms of mandibles of puppies of the second group. A, Control dog. B, Dog 7 after 74 days peroral ingestion of about 0.2 Gm. sodium fluoride daily. Dense periosteal bone deposition. Disturbance of calcification of the buds of the permanent teeth is perceptible.

The radiographic examination of the bones of the fluorine dogs revealed a systemic involvement of all bones, with a distinct predilection for the long bones, jaws, and vertebrae (Figs. 1 and 2). There was a thickening due to massive periosteal bone formation, superimposed upon the old cortex. A fairly sharp boundary against the compact bone was preserved. The new periosteal layer on all bones appeared extremely wide and was particularly pronounced at the insertion of muscles. While the contours of the thickened bones of the dogs of the first group and of the mother dog of the third group were sharp, the bones of the fluorine puppies showed blurred outlines. The spongy part of their bones revealed a distinct osteoporosis (Fig. 3). The medullary cavities of the bones of the mother dog appeared narrowed by endosteal osteosclerosis.

TABLE I

| DOG | DIET | DAILY SUPPLEMENT OF SODIUM FLUORIDE IN BASAL RATION (GRAMS) | AGE AT BEGINNING OF EXPERIMENT (DAYS) | WEIGHT AT BEGINNING OF EXPERIMENT (GRAMS) | AGE AT DEATH (DAYS) | DAYS ON DIET | WEIGHT AT DEATH (GRAMS) | |
|-----------|--|---|---------------------------------------|---|---------------------|--------------|-------------------------|---|
| 1 Male | Control | 0 | 98 | 4,500 | 194 | Control | 14,500 | Group 1 |
| 2 Male | Normal food; rye bread, meat, milk; later plenty of bones | Control | | | | | | |
| 3 Male | Normal food | 0.2 | 98 | 5,000 | 194 | 96 | 10,500 | |
| 4 Male | Normal food | 0.1 | 98 | 5,500 | 194 | 96 | 12,500 | |
| 5 Male | Normal food | 0.1 | 98 | 5,000 | 194 | 96 | 11,700 | |
| 6 Male | Normal food and cod-liver oil and salt mixture of Osborne and Mendel | 0.1-0.2 | 47 | 3,400 | 117 | 70 | 6,300 | |
| 7 Female | Same as No. 5 | 0.1-0.2 | 47 | 3,150 | 122 | | 5,400 | Group 2 |
| 8 Female | Same as No. 5 | 0.1-0.2 | 47 | 2,700 | 121 | 74 | 5,000 | |
| 9 Female | Normal food and cod-liver oil and salt mixture of Osborne and Mendel | Control | 47 | 2,700 | 122 | Control | 8,700 | |
| 10 Male | Suckled by fluorine-poisoned mother dog No. 9 | 0.4 | 330 | 11,500 | 670 | 340 | 14,500 | Had a litter of 7, following 156 days of sod. fluor. administration. Received 62.4 Gm. of sod. fluor. throughout the 156 days |
| 11 Female | Suckled by fluorine-poisoned mother dog No. 9 | | | | 28 | | 1,350 | |
| 12 Female | Suckled by dog No. 9 for 28 days, then fed normal food | 0.1 | 28 | 1,300 | 156 | 128 | 3,300 | Group 3 |
| 13 Female | Same as No. 12 | 0.1 | 28 | 1,400 | 57 | 29 | 1,730 | |
| 14 Male | Same as No. 12 | 0.1 | 28 | 1,300 | 95 | 67 | 2,500 | |
| 15 Male | Same as No. 12 | 0.1 | 28 | 1,300 | 74 | 46 | 2,700 | |
| 16 Male | Same as No. 12. Control | Control | 28 | 1,100 | 156 | Control | 8,000 | |

Among the bones most involved by periosteal thickening and endosteal osteoporosis the jaws occupied a prominent place (Fig. 2). The radiographic examination of the jaws which contained the buds of the permanent teeth demonstrated, besides the bone changes, a very distinct change of the hard tissue of the tooth buds as compared with the control.

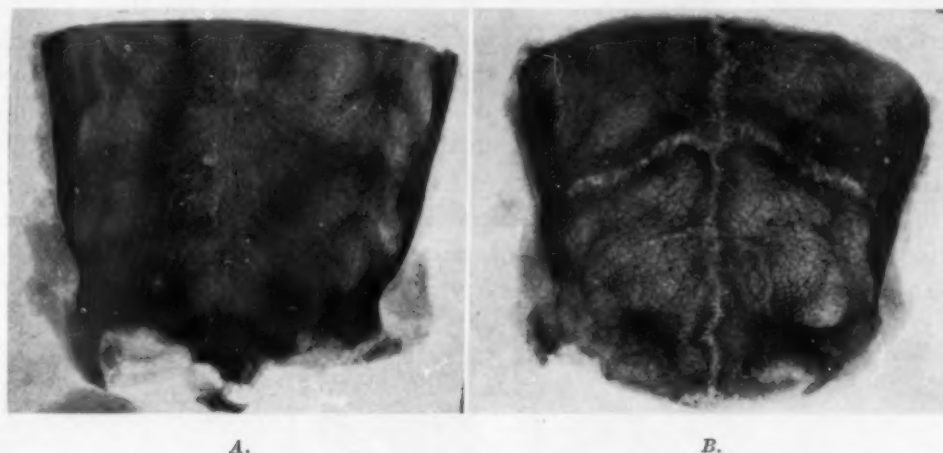


Fig. 3.—Roentgenograms of skull bones of puppies of the third group. *A*, Control dog. *B*, Dog 12 after 100 days peroral ingestion of about 0.1 Gm. sodium fluoride daily. Osteoporosis, sutures widely opened.

The gross examination of the bones of the fluorine dogs revealed a striking thickening. Their surfaces appeared rough, uneven, and chalky. The cut surfaces of the bones which were sawed in two in the axis showed that the thickening was due to an excessive periosteal bone formation, while the original cortex was thin. The consistency of these thickened bones differed remarkably. The bones of the mother dog (No. 9) were extremely hard, those of the first group were rather brittle, those of the second group could be, with some difficulty, cut with a knife, whereas the bones of the puppies of the third group were so soft that they were bent and cut easily.

The eruption of the permanent teeth was considerably delayed in all dogs on fluorine diet.

MICROSCOPIC EXAMINATION OF BONES AND TEETH

Group 1.—The outstanding feature was the contrast between the extremely broad layer of the sclerotic periosteal bone and the atrophic spongy bone (Fig. 4). The trabeculae of the latter were sparse and slender. They consisted of newly formed bone. A moderate number of them contained a core of old bone outlined with resorption lines. They were covered with narrow osteoid zones that appeared coated with a dense layer of high, columnar osteoblasts. Osteoclastic activity was almost absent, thus indicating that the stage of resorption that preceded the present stage, and thinned the spongy trabeculae and compact bone, had been succeeded by deposition of bone. This has been evidenced also by the thickening of the previously thinned cortex. The cortex of the bones and, mainly, of the mandibles showed that new layers of dense bone (Figs. 4 and 5) were deposited from inward on the resorption lines. The boundary between the compact bone and the sclerotic periosteal layer was fairly well preserved here and there, but occasionally not discernible (Fig. 4). The calcification of the spongy, compact, and periosteal bone did not appear interfered with in the dogs of this group, which received only 0.1 Gm. of

sodium fluoride daily, but appeared altered in the dog with 0.2 Gm. per day. Here, the central areas of the trabeculae of the broad periosteal bone contained more or less dense accumulation of very fine granules that took the hematoxylin stain. These granules did not tend to fuse together, and were not noted in the peripheral part of the newly deposited periosteal bone, which remained uncalcified and stood out clearly against the granular layer. While these granules decreased very remarkably in number in the spongy bone, they increased in size. Here the substance of which the granules consisted filled some of the osteocytes and also occupied the lacunae and canaliculi (Fig. 6). Yet deep bluish-staining spots were observed also lying freely in the bone matrix and between the cells of the bone marrow. Some of the small Haversian systems and some capillaries of the bone marrow were stocked with this substance. The osteoblasts, though surrounded by this dark-staining substance, remained free, while few osteoclasts contained aggregations of this material.



Fig. 4.



Fig. 5.

Fig. 4.—Dog 3. Sodium fluoride administered for 96 days. Buccolingual section through mandible with the permanent first premolar. Tremendous sclerotic periosteal bone layer. Osteoporosis of the original jawbone.

Fig. 5.—Higher magnification of the dense sclerotic periosteal bone of Fig. 4.

The bone marrow of the spongy bone was hyperemic but otherwise unaltered. The narrow marrow spaces of the periosteal bone contained a fibrous tissue with dilated congested capillaries. The costochondral junction of the ribs and the epiphyseal cartilage showed normal conditions.

The permanent teeth of the dogs of this group had erupted. All teeth of the fluorine-poisoned dogs, but especially the premolars and molars, had brownish, discolored, corroded, enamel hypoplasias around the cusps (Fig. 7). The enamel in this area was chalky and could be easily chipped off.

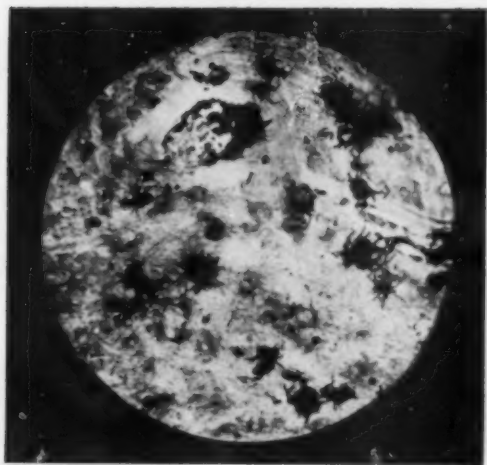
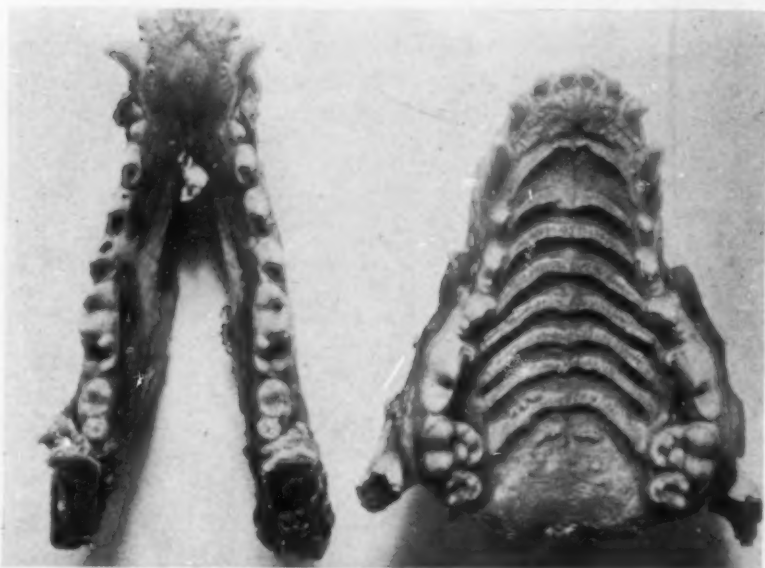
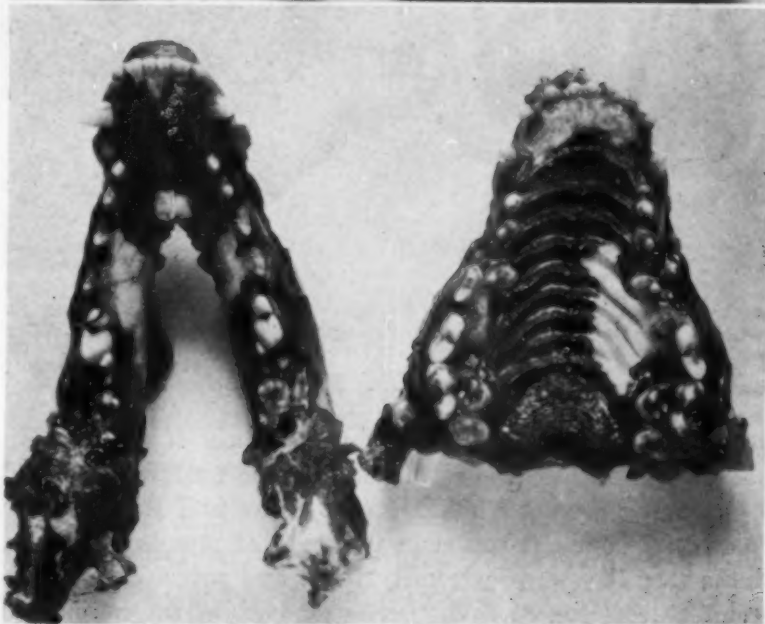


Fig. 6.—Bone cells filled with deeply staining material.



A.



B.

Fig. 7.—Occlusal view of the jaws of dogs of the first group. A, Control dog. B, Dog 3 after 96 days administration of sodium fluoride. Note discolored hypoplasias of the premolars and molars.

Since the enamel had been dissolved by the calcification of the specimen, only remains of it in connection with the epithelial attachment were preserved in the sections. These small narrow islands of enamel were characterized by many distinct incremental lines and a coarse irregular calcification of the area next to the epithelial attachment. Inclusions of cellular elements were observed in some sections.

The dentine of the erupted teeth showed an excessive stratification. Its calcification remained globular throughout and many a capillary, sometimes extended from the pulp to the dentinoenamel junction and also to the dentinocemental junction, was entrapped in the dentine. The dentinoid was unusually wide and coated with a layer of fairly well-preserved odontoblasts which occasionally revealed hydropic degeneration. The pulp tissue appeared unchanged with the exception of a moderate hyperemia. The primary and secondary cementum was normal. The cementoid zone was of normal width, thus contrasting with the dentinoid layer.



Fig. 8.—Buccolingual sections through the upper jaws of dogs 8 and 5 showing the deciduous tooth and the bud of the permanent tooth. A, Control dog. B, After 117 days fluorine administration combined with vitamin D. Tremendous thickening of the jaw by new periosteal bone, osteoporosis of the spongy part. Note the Owen's lines.

Group 2.—The microscopic feature of the bones of these dogs, which received an additional antirachitic supplement to the food, differed quite remarkably from the first group. While the extremities and the skull bone showed moderate periosteal bone deposition in the areas of muscle insertions, the mandibles and the maxillas were extremely thick and revealed a complete change of their architecture (Fig. 8). The periosteal bone of the jaws and long bones was composed of trabeculae perpendicularly arranged to the horizontal axis and not as densely packed as in the first group. The cortex appeared very ir-

regularly resorbed by osteoclasts (Fig. 9). The calcification of all newly formed bone was very irregular and spotty; however, the osteoid layers, though occasionally quite broad, were not as thick as true rachitic osteoid. The bone marrow contained many extremely dilated vessels and appeared fibrous and even gelatinous. The spongy trabeculae of the long bones were coated with osteoclasts, and osteoclasts were very active also on the periosteal surface of the cortex but outside from the muscle insertion, so that a thinning of these parts of the bones resulted. The resorption was very noticeable next to the growth centers, such as epiphyseal regions and costochondral junctions.

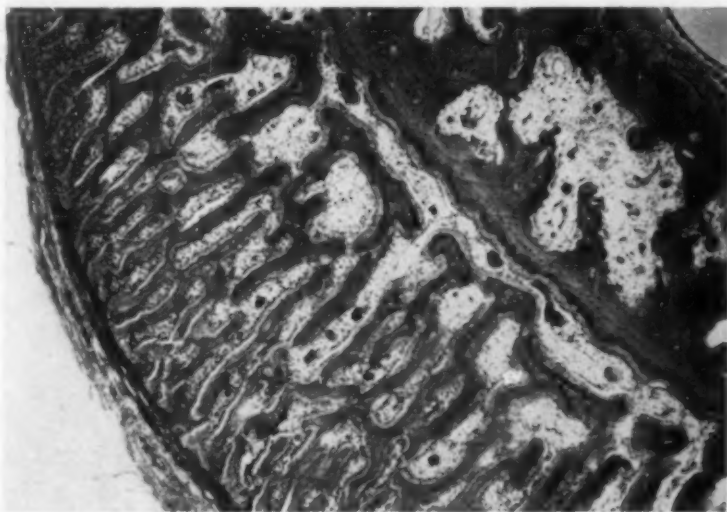


Fig. 9.—Periosteal bone trabeculae perpendicularly arranged upon the osteoclastically eroded cortex of the femur of Dog 5.

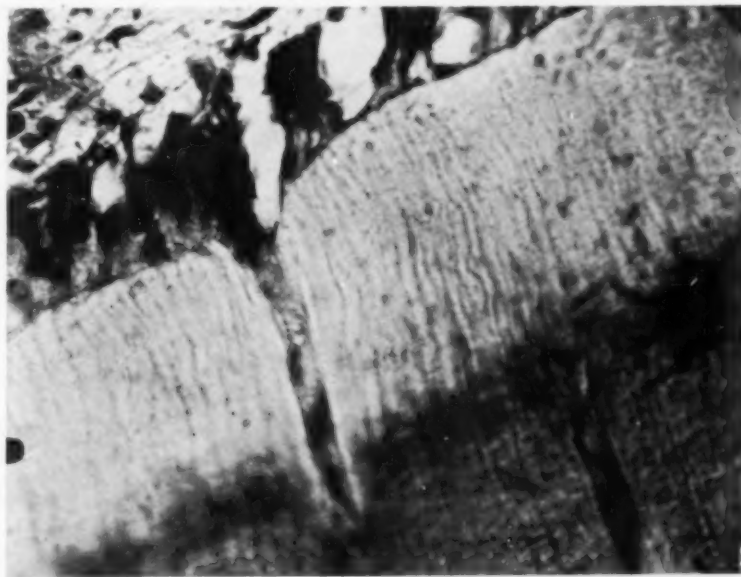


Fig. 10.—Dog 6. Capillaries with cells entrapped in the predentine and dentine.

Teeth.—Some of the deciduous teeth were still in position and their histologic feature did not differ from the erupted teeth of the first group.

The buds of the permanent teeth, of course, were more affected. The dentine was of normal thickness, yet the disturbance of the calcification brought about an extraordinary pattern of Owen's lines of varying width. Numerous

vessel and cell inclusions were seen in the dentine (Fig. 10). The predentine was not too extremely broad. The pulps showed dilated congested vessels. The enamel of the tooth buds, some of which had advanced close to the gingiva, was represented by an astounding narrow strip of enamel, which was covered by a layer of pleomorphic ameloblasts, in connection with proliferated, stratified epithelium. Upon the surface of this thin line of enamel there were sporadically superimposed islands of an amorphous substance, which extended into the epithelium and appeared coated by more or less preserved ameloblasts (Fig. 11).

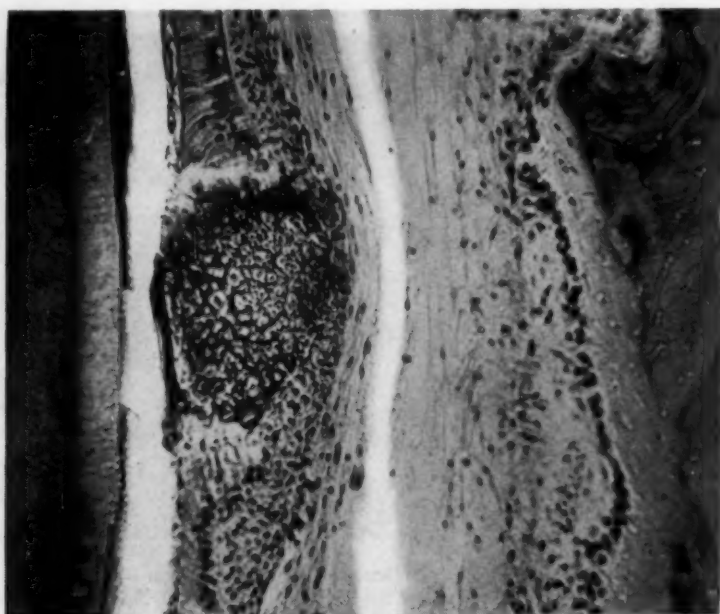


Fig. 11.—Dog 5. Isolated area of abortive enamel upon thin layer of normal enamel extending into the outer epithelium.



Fig. 12.—Dog 7. Ameloblast layer and outer epithelium lifted from the thin layer of enamel.

These small areas occasionally exhibited the characteristics of an atypical enamel, but more often a structureless, homogenous mass, containing sparse, bluish-black globules. There were also small round hyaline-like areas in the epithelium surrounded by a ring of calcified tissue. Some of the epithelial cells appeared calcified. Occasionally, the enamel epithelium appeared to be detached from the strip of enamel by threads and nuclei, which were remains of destroyed cells (Fig. 12).

The tooth sac tissue showed hyperemic vessels and an occasional fibrosis.

Group 3.—The discussion of the findings in this group will be divided into three parts; the changes of the bones and teeth of the two puppies (Nos. 10 and 11) which were suckled by their fluorine-poisoned mother before they were killed, then those of the four puppies (Nos. 12, 13, 14, 15) which were fed mother's milk for four weeks and afterward received fluorine-containing food, and finally the changes of bones of the mother dog.

Puppies 10 and 11.—The long bones and the jaws exhibited remarkable periosteal bone deposition which developed particularly at the insertion of muscles and tendons while osteoclasts resorbed the cortex from inside. The calcification was undisturbed and the osteoid zones did not exceed the normal width (Fig. 13). The costochondral junctions and the other bones were normal.

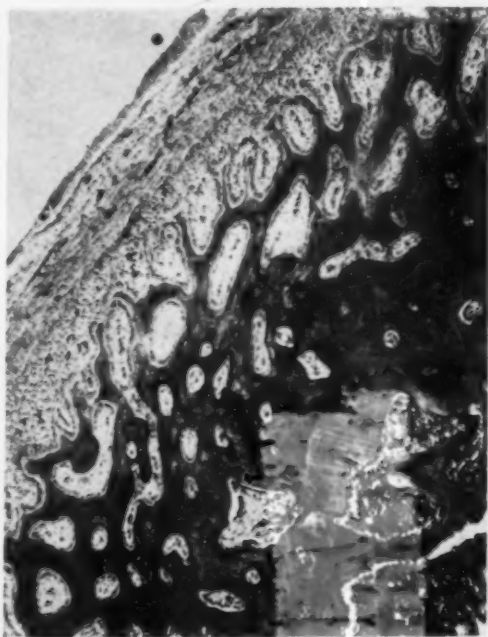


Fig. 13.—Dog 10. Fed by milk of fluorine-poisoned mother dog. Periosteal bone deposition with broad osteoid zones.

No changes could be found in the tooth buds of those two puppies which received only the fluorine-containing mother's milk. The prenatal and post-natal enamel and dentine were unchanged.

Puppies 12, 13, 14, and 15.—All the bones of Puppies 13, 14, and 15 were considerably soft in spite of the thick layers of new periosteal bone which was laid down in a radiating fashion upon a cortex that appeared resorbed from inward. These periosteal bone trabeculae were densely packed and had extremely broad osteoid zones which were coated with a layer of abnormally large osteoblasts. The central parts of these trabeculae showed globular spotty calcification.

The fibrous marrow of this area contained abundant dilated capillaries. Numerable osteoclasts were observed on the trabeculae of the spongy bone while the osteoblastic activity was very moderate. The appearance of extremely large bone marrow spaces evidenced that many trabeculae had been completely resorbed and marrow spaces had fused together. The marrow in the areas of intense osteoclastic activity consisted of fibrous, sometimes gelatinous, tissue

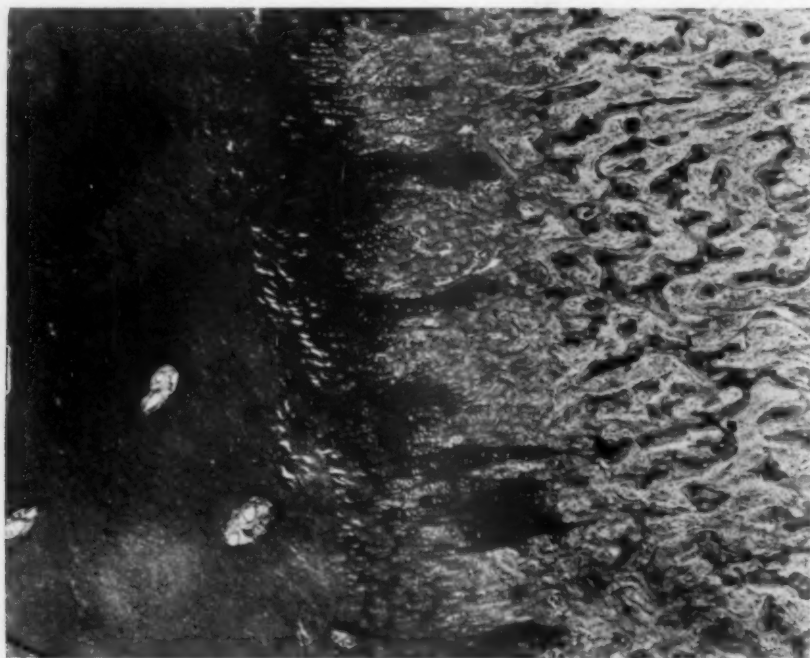


Fig. 14.—Dog 15. Ricketslike disturbance of the costochondral junction of the rib.

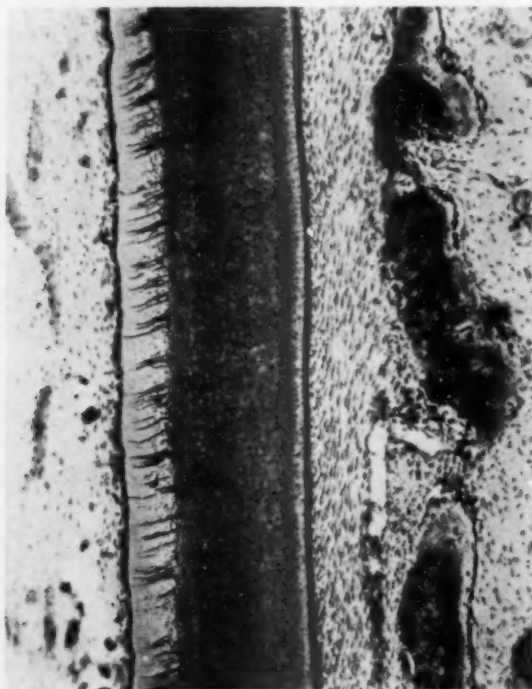


Fig. 15.—Dog 15. Area of the root, periodontal membrane, and alveolar bone of a deciduous tooth. Broad osteoid and dentinoid zones. Appearance of incremental lines in the dentine blurred by deeply staining coarse granules scattered throughout and grouped in radiating threads in the predentine.

with hyperemic vessels. The ossification zones of the ribs were irregular and showed small isolated islands of calcification in the otherwise hyperplastic zones of proliferating cartilage (Fig. 14). The epiphyseal lines appeared very wide and irregular.

The microscopic picture of the bones of Puppy 12, which received the fluorine supplement for 128 days, differed somewhat from the other puppies of the same group. Although the periosteal bone deposition was equally voluminous, and abnormally broad osteoid zones surrounded the trabeculae, the spongy bone appeared dense. There was considerable new endosteal bone, the trabeculae of which were irregularly calcified and covered with broad, not yet calcified layers. Little osteoclastic activity was observed.

The deciduous teeth of these puppies revealed an uniformly calcified prenatal dentine. The postnatal dentine, however, showed very accentuated Owen's lines. Layers with poor, yet globular, calcification alternated with broad layers of uncalcified dentine. Black, round granules scattered through the postnatal dentine and packed chainlike in the tubules of the wide predentine were an outstanding finding (Fig. 15). Not less striking was a definite hypercementosis composed of very poorly calcified and, here and there, fully uncalcified secondary cementum. No changes except a hyperemia were noticed in the pulps.

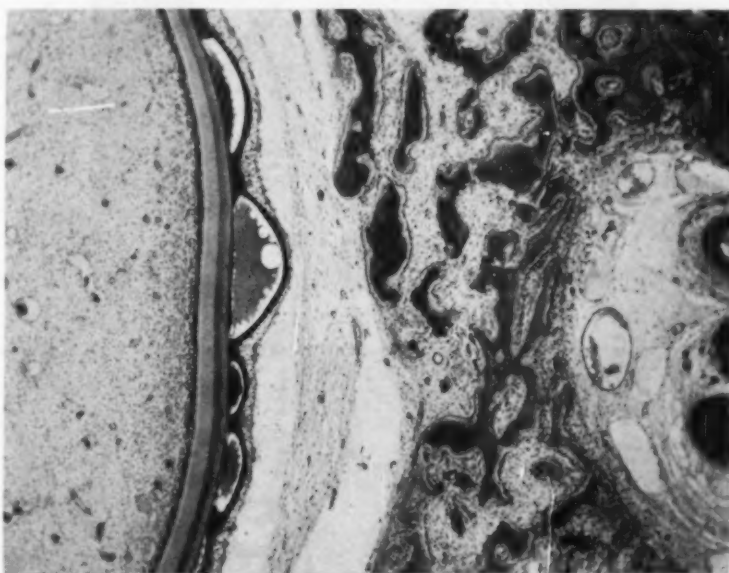


Fig. 16.—Dog 15. Area of the crown of a bud of a permanent tooth with tooth sac and bony crypt. Broad predentine zone. Four blisterlike elevations of the united enamel epithelium containing a structureless substance with very fine deeply staining granules. Wide osteoid zones on the newly formed trabeculae of the bony crypt.

The buds of the permanent teeth were affected very much. The thinness of the enamel layer contrasted very noticeably with the thickness of the dentine in all tooth buds. The narrow layers of enamel were coated with the united epithelium; in some areas the stellate reticulum was still present. In many points of the enamel surface the united epithelium appeared to be detached. Amorphous accumulations of enamel with cell inclusions were observed there (Fig. 16). Black granules were unevenly distributed in the ameloblasts, while the cells of the intermediate and outer layer remained free. The prenatal dentine was well-calcified, while the postnatal layers showed severe disturbances

of calcification and occasionally an abundance of black granules (Figs. 17 and 18). The pulps were normal and no granules were noted in the odontoblast.

Mother Dog.—The long bones, the vertebrae, and the jaws showed formation of extremely well-calcified, sclerotic periosteal and endosteal bone, in the areas of muscle insertion. The marrow in this part appeared to be fibrous. It is obvious that no tooth changes were observed.

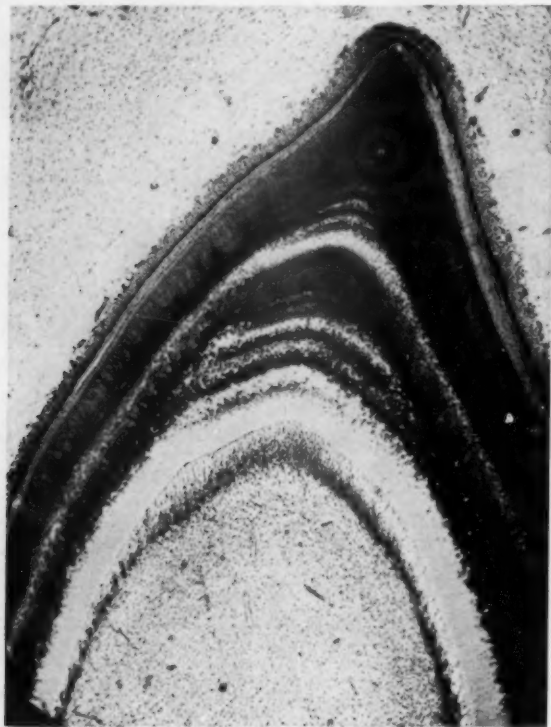


Fig. 17.—Dog 15. Area of the crown of a bud of a permanent tooth. Extremely thin line of enamel, excessive amount of dentine which shows a well-calcified prenatal layer but a granular deficient calcification of the broad incremental layers and an extremely wide predentine.

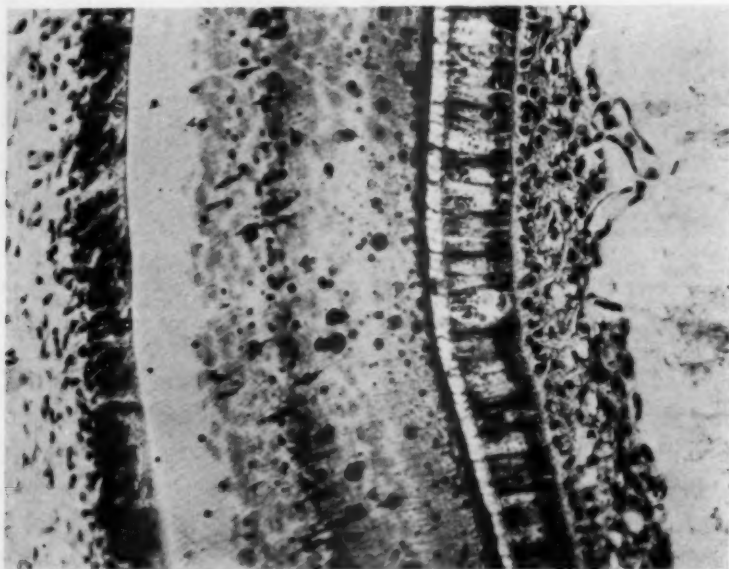


Fig. 18.—Dog 14. Area of the crown of the bud of the lower second permanent premolar. Thick layers of very poorly calcified dentine with broad predentine zone and coarse deeply staining granules. Very narrow layer of enamel coated with united epithelium. Ameloblasts contain granules mainly in the proximal end of their cytoplasm. Degenerative changes of ameloblasts.

DISCUSSION

Chronic fluorine poisoning produced not only the well-known changes in the enamel and dentine of developing teeth, but also a systemic involvement of the bones of dogs. The effect depended on the age of the animals, the daily dose, and the length of the time of fluorine administration. The earliest changes of the bones were observed in puppies exclusively fed the milk of their chronically fluorine-poisoned mother dog for twenty-eight days. It is particularly interesting that this periosteal bone deposition occurred prior to any alteration of the tooth buds. It can be concluded from this observation that the cells of the periosteum were stimulated to assume osteoblastic function even before the enamel-forming cells were affected. It is difficult to reconcile this fact with the repeated statements, verified by experiments with rats, that the earliest changes of chronic fluorine intoxication are found in the enamel and dentine.

It is the excessive periosteal bone layers that were seen in all dogs used in the experiment. The density of these trabeculae, laid down perpendicular to the longitudinal axis of the bones, varied according to the duration and intensity of the fluorine action. Small doses of fluorine administered over a long time and started at an early age resulted in a very dense periosteal bone formation.

The periosteal bone deposition appeared to be growing rapidly in young animals but its development was slow and limited in the old dog in spite of the long duration of fluorine administration. This indicated that the growing bones reacted faster and more intensely than the fully developed ones. The matrix of the periosteal and endosteal bone did not seem altered; only the osteocytes were larger and irregularly arranged.

The thickening of the bones was restricted to the insertion of muscles and tendons. While the deposition of the periosteal layers mostly occurred in certain areas, the osteoclastic resorption, mainly evident in the young dogs, was diffuse, so that there were also osteoporotic parts lacking any remarkable periosteal bone. Therefore, the development of the new periosteal bone can hardly be conceived to be a compensatory reaction to the osteoclastic resorption of the spongy trabeculae and the inner surface of the cortex. Similar periosteal apposition is seen in experimental and human rickets and osteomalacia but never reaches the extent as in chronic fluorine intoxication.

The osteoclastic resorption of the spongy and compact bone, particularly pronounced in the young dogs, was always lagging behind the osteoblastic activity of the periosteum, and gave way in the older dogs to endosteal bone deposition. This thickening of the spongy bone of older animals was associated with the thickening of the cortex.

As a whole, the costochondral junctions and the epiphyseal lines did not reveal noticeable alterations, with the exception of Puppies 12 and 15, which were fed fluorine-containing mother milk and afterward received normal food, with a fluorine supplement. It was only in these cases that the excessive proliferation of cartilage cells, the vascularization of the cartilage, and the thickening and irregularity of the epiphyseal line resembled the changes in rickets.

In discussing the very obvious alteration of the calcification of the mesodermal tissues (bone, dentine, cementum) in experimental chronic fluorosis, one faces many difficulties, as important details of the normal calcification are still a mystery. The disturbances of calcification were dependent upon the age of the dogs and the dose of fluorine, but the incorporation of an antirachitic supplement into the fluorine diet did not prevent the development of changes in bones and teeth due to the fluorine. The younger the dog and the greater

the dose of fluorine, the more pronounced the impairment of the calcification. In some of the dogs, deep blue-staining globules of varying size were noted in the bone marrow. These were diffusely scattered between the cells, but they were also seen aggregated in capillaries and in a few osteoclasts. They were mainly found in the substance of the bone trabeculae and in the osteocytes. The fact that these spots failed to appear in the very recently laid down, not yet calcified, periosteal and endosteal layers points to their nature as a bone-salt compound. Deposition of coarse granules of the same nature were seen by Roholm² in the bones of cryolite workers. It should be emphasized that these granular deposits were observed in only a few of the puppies, however, and not in all sections of the same bone. This finding may indicate that only a part of the calcium salts combined with the fluorine to form calcium fluoride, which I consider the dark lumps and spots to be. While Schour and Smith,¹² who observed the granules in the ameloblasts, and Oehnell and co-workers¹³ regard these irregular granules as calcium fluoride, Roholm² holds that they are the result of a precipitation of the mineral salts due to the detrimental effect of fluorine on the enzymatic processes that play an important role in the calcification of bone and tooth tissues.

The osteoid zones were somewhat wider than normal but reached the abnormal width of rachitic osteoid only in the bones of Puppies 12 and 15. It was in these young animals that the costochondral junctions and the epiphyseal lines showed changes characteristic to rickets. The interference with the normal calcification, associated with an extreme periosteal bone formation, was very noticeable in the growing animals, decreased with the advancing age, and was substituted by an excessive calcification in adult dogs.

The sufficient calcification of the new periosteal and endosteal bone of the older dogs and the mother dog led to an osteosclerosis as seen by Sutro¹¹ in rats with chronic fluorine intoxication. Sutro thought that these bone trabeculae resemble the structure of Paget bone. Wolff and Bauer,¹⁴ however, proved that cranial bones of six Paget cases contained less fluorine, by far, than those of normal individuals. The author considers the architecture of the sclerotic bone in chronic fluorine intoxication essentially different from the osteosclerosis in Paget's disease because of the absence of the typical "mosaic" pattern of the Paget bone, brought about by rapidly alternating resorption and deposition.

The transformation of the bone marrow into a hyperemic fibrous tissue of various density, containing hemorrhages and gelatinous areas, is the usual reaction to disturbances of the calcium-phosphorus metabolism.

It appears from the foregoing observations that the bone changes of only the young dogs somewhat resembled rickets.

The dentine of the tooth buds and those erupted teeth which were affected by chronic fluorine intoxication during their development appeared thicker than normal and exhibited broad Owen's lines, with coarse globular calcification, very wide predentine zone, and a startling number of entrapped capillaries, thus revealing the essential feature of dentine in rickets. These alterations of the dentine remained unchanged throughout, while the trabeculae of the new periosteal and endosteal bone, that were originally also very poorly calcified, became well, yet irregularly, calcified.

As in the bones of some of the puppies, aggregations of deeply staining granules were found to fill the tubules of the predentine, and to be scattered all over the irregularly calcified dentine. However, neither the odontoblasts nor other pulp cells revealed those small, deeply staining granules that were

seen in the ameloblasts and stratum intermedium. While the changes of the dentine, with the exception of the occasional appearance of deeply staining deposits of calcium fluoride, were of rachitic type, the involvement of the enamel-forming cells differed fundamentally from the development of hypoplasias, as described by the author¹⁵ in human and experimental rickets.

In contrast to the increased amount of dentinal ground substance, there was a very noticeable decrease of the amount of enamel in the tooth buds of the experimental puppies, compared with the control animals. An uneven thin layer of enamel with accentuated incremental layers covered the coronal dentine of the tooth buds, and upon its surface irregularly shaped islands of rudimentary or amorphous enamel were occasionally found. Some of these abortive enamel bodies contained deformed, sometimes calcified, epithelial cells. The enamel epithelium exhibited a variety of changes particularly involving the ameloblasts. There were normal ameloblasts but their arrangement was disturbed by spaces between them, or their size and shape appeared altered. Even a complete absence of enamel epithelium confined to smaller areas was noted. Here and there the enamel epithelium was lifted from the enamel surface by the interposition of abortive enamel. Calcification of degenerated ameloblasts and cells of the stratum intermedium was a common finding. Globules of a more or less amorphous material were also observed in the stratum intermedium, and, being separated from the ameloblast layer, they must be conceived to be of a secretion product of the cells of the stratum intermedium. The author¹⁵ also described these considerably large droplets in his studies of enamel hypoplasias in rickets, and other systemic interferences with the calcium-phosphorus metabolism. Recently, Wassermann¹⁶ demonstrated this secretion of globular material by the cells of the stratum intermedium and thinks that these droplets and the fine granules secreted by the ameloblasts in the earliest stage of enamel formation are identical. In those sections that showed deeply staining granules in the bone and dentine, the enamel epithelium and the defective enamel also exhibited these "calcium fluoride" bodies. Schour and Smith discussed the probability that this calcium fluoride, taken up by the enamel- and dentine-forming cells, may act as a foreign body and disturb the function of these cells. However, these inclusions were seen in a few of the animals, while others were entirely free of them yet exhibited the same alterations of dentine, enamel, and enamel epithelium.

The cementum of the erupting and erupted teeth of the dogs showed changes identical with those of the bones.

It is of interest to stress briefly the difference of the histologic feature of the damage to the enamel epithelium in rickets and in chronic fluorosis. In his study on "enamel hypoplasia," the author¹⁵ pointed out the fundamentally different action of rickets on the odontoblasts and ameloblasts. In human and experimental rickets, odontoblasts and osteoblasts are involved alike. The hard substances (dentine and bone) are laid down by these cells in abundance but remain uncalcified for the duration of the disease. The enamel epithelium, however, is affected here and there by a transudate that originates in the highly vascular tooth sac and penetrates through the enamel epithelium, thus detaching parts of the ameloblasts in a blisterlike fashion from the enamel. Pressure atrophy of this area of enamel epithelium and hydropic degeneration of the ameloblasts are the results leading to the more or less abrupt and local discontinuation of enamel formation. The author advanced the thought that, in spite of the disturbance of the calcium-phosphorus metabolism, the ameloblasts due to their particular avidity for calcium-phosphorus are able to obtain enough mineral

salts to secure a calcification of the enamel in the areas not greatly affected even in rickets. This opinion of the author seems supported by the histochemical studies of Hintzsche and Baumann¹⁷ who found calcium, phosphorus, and potassium particularly accumulated in the ameloblasts. In experimental chronic fluorosis, however, fluorine acts as "a general protoplasmic and enzymatic poison (De Eds¹⁸). According to Phillips and Hart,¹⁹ it inhibits enzymic systems and disturbs the actively metabolizing systems involving phosphoric acid esters, thereby interfering with the calcification.

The author supposes that it is difficult to explain the contrasting effect of fluorine upon the enamel on one side and dentine on the other side. To place the emphasis on the different germinal origin of the cells producing these two substances is not fully satisfactory. Perhaps Hampp's²⁰ study on the mineral distribution in developing teeth offers a better clue to this question. Hampp proved that the fully differentiated ameloblast just prior to the formation of enamel reveals large quantities of calcium and magnesium in its cytoplasm and confirmed thus the work of Hintzsche and Baumann.¹⁷ Hampp stated further that the odontoblast seems to be constant in its mineral content because it is fully differentiated in a very early stage and plays only a passive role in dentine formation. If one applies this thought to the problem in question, one becomes inclined to believe that in processes associated with impairment of calcification the formation of dentinal matrix can continue, while the enamel formation discontinues due to the gradual destruction of the ameloblasts. The presence of the deeply staining granular bodies (calcium fluoride?) in the ameloblasts, and their constant absence in the odontoblasts may bear some significance and help to explain the gradual destruction of the former and the preservation of the latter.

It may be difficult to explain the startling bone involvement associated with "mottled enamel," as revealed in this study, with the relatively large amount of sodium fluoride administered daily to the dogs, since the very little amount of fluorine, which has been transferred with the milk of the mother dog to the puppies, produced bone but no tooth changes.

The studies of Shortt and co-workers,⁵ Pandit and co-workers⁶ in India, Oekerse⁷ in South Africa, Linsman and McMurray⁸ in this country, and particularly Kemp, Murray, and Wilson⁹ in England are of significance. These authors reported coinciding involvement of teeth and bones in human fluorosis. It is from this point of view that careful roentgenographic studies of the skeleton of children living in fluorosis areas and exhibiting mottled enamel seem to be highly indicated. Since the bones are subjected to a constant rebuilding process in which, normal conditions presumed, deposition and resorption are well balanced, it can be assumed that most of the bone changes in fluorosis of children will disappear as time goes on, unless they are too excessive. However, the disturbances about the epiphyseal lines of the bones involved may persist and give rise to various processes.

SUMMARY

Chronic fluorine intoxication of puppies produced extensive systemic changes of the bones and developing teeth. The intensity depended upon the age of the animal, the dose, and the duration of the administration of sodium fluoride. In puppies fed exclusively the milk of their fluorine-poisoned mother, changes of the bones were observed prior to those of the tooth buds.

The fact that in experimental fluorosis the changes of bones and developing teeth could not be prevented nor alleviated by an antirachitic diet, and calcification of a startling amount of periosteal and endosteal bone occurred in spite of continued administration of fluorine suggests the idea that the pathogenesis of the bone and tooth lesions in rickets and fluorosis differs.

Of course, the enzymatic and protoplasmic action of fluorine produced ricketslike changes in the bones and the dentine of puppies, but its effect on the bones of the older dogs and the enamel in general differed from rickets.

These experimental studies and recent observations of dental fluorosis in children and adults associated with bone changes indicate the necessity of a thorough roentgenographic examination of the skeleton of children showing "mottled enamel" in fluorine areas in this country.

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CHEMOTHERAPY IN ORAL SURGERY

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GOOD therapeutic results in oral surgery are dependent on more than the mere application of a sulfonamide into a wound as a prophylactic or therapeutic agent. It is evident that the sulfonamides have relegated many other valuable agents heretofore used by the oral surgeon to the background, but this should not mean that the oral and topical use of sulfonamides should displace sound surgical procedure.

The final result of any operative procedure is influenced by the surgeon's skill, and unless this skill is utilized to its fullest extent, failure cannot be placed with anyone but the surgeon. It cannot be stressed often enough, nor strongly enough, that the sulfonamides do not represent a panacea, but are only adjuncts to sound surgical practice.

The choice of the proper sulfonamide to be used when such use is indicated was simplified by Long¹ who, after a comprehensive review of the literature, concluded: "From all points of view, sulfanilamide seems at the present time to be the drug of choice for topical application." It has definitely been shown that sulfanilamide has a greater power of concentration and diffusibility than the other sulfonamides, and it also has the advantages of being absorbed more readily.^{2, 3}

The successful local application of powdered sulfanilamide following dental and oral operations was reported by Sinclair.^{4, 5} He found that the implantation of the drug in tooth sockets or in wounds after dental operations, where infection had occurred or was anticipated, generally reduced infection, facilitated healing, and prevented or controlled postoperative pain. Furthermore, roentgenograms of the sockets showed a normal filling in bone substance. The conclusion drawn was that sulfanilamide hastens healing by controlling infection.

In a series of one hundred patients whose teeth had been extracted because of the presence of advanced pyorrhea, Lanier⁶ reported a marked decrease in the usual after-extraction soreness, pain, and dryness of sockets, following the local application of sulfanilamide.

That the local application of sulfanilamide into sockets following tooth extracts prevented dental infections was claimed by Norris, et al.⁷ A method for the local application of sulfanilamide in root canal therapy was described by Adams.⁸ The canal was irrigated with a hot (about 140° F.) suspension of sulfanilamide by means of a syringe. Claim was made that sterility was readily established, and sulfanilamide appeared to be more effective than the other sulfonamides.

Literature is replete with references to the successful use of sulfanilamide in compound fractures. Hendrick⁹ used powdered sulfanilamide locally in compound fractures as well as in potentially infected wounds, and was convinced of its efficacy in reducing serious infection. In the treatment of com-

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pound fractures of the mandible, gratifying reduction in infection was obtained by the use of sulfanilamide locally, according to Sinclair.⁴ The reduction of the incidence of infection in compound fractures from 27 per cent to 5 per cent by the local implantation of sulfanilamide was reported by Jensen, et al.¹⁰

Our experience with sulfonamides used topically has demonstrated that more than a mere dusting of the lesion was necessary to obtain the desired therapeutic effect. When a sufficient amount of the sulfonamide was used, "caking" usually occurred, and this in many cases produced the effect of a foreign body. It was not possible to remove the "cake" by gentle flushing; it was often necessary to pry the caked powder out of the lesion. Furthermore, where this phenomena did not occur, we found that a prolonged use of sulfonamide powder would retard healing. This was manifested by dull-appearing sluggish tissue, and the absence of healthy granulations.

Veal and Klepser¹¹ reported similar experiences, but found a means of using sulfanilamide topically without the attendant disadvantages we experienced. They used allantoin 2 per cent, sulfanilamide 10 per cent in a water-miscible base which permitted the release of the active components: sulfanilamide to exert a bacteriostatic action, and allantoin to stimulate healthy granulations. The base for this ointment contained triethanolamine, glycerin, stearic acid, and water.*

Allantoin as a cell proliferant was first reported by Macalister¹² who used extracts of comfrey root to treat chronic ulcers which had failed to heal under all other types of treatment. The results were exceedingly good and he was convinced of the efficacy of allantoin.

Robinson,¹³ investigating the reason for the excellent results obtained in maggot therapy of osteomyelitis as suggested by Baer,¹⁴ discovered allantoin to be present in the excretions of the maggots, and that the allantoin was responsible for the healing action.

The successful use of allantoin was reported by Kaplan,¹⁵ who concluded: "Allantoin induces healing by stimulating healthy granulations and removing necrotic material; treatment is painless; it acts locally as long as allantoin is in contact with the wound."

The successful treatment of osteomyelitis of the mandible was reported by Gordon.¹⁶ Treatment consisted of hourly irrigations with allantoin; healing set in, the entire treatment was ambulatory, and proved entirely beneficial to the patient. Sussman¹⁷ reported on the use of allantoin in osteomyelitis of the mandible and fracture of the jaw. He concluded: "It seems to us that in allantoin we have another drug which can be added to our armamentarium for the treatment of suppurative conditions. It appears to stimulate the formation of granulation tissue and reduce the time element in the healing of these conditions."

The allantoin-sulfanilamide ointment in a water-miscible, nongreasy base was used as an adjunct to oral surgery in 184 patients with the following conditions: 27 gingivectomies, 2 compound fractures of the jaw, 2 osteomyelitis of the mandible, 15 cases of maxillary sinus pathology of dental origin, 7 Caldwell-Lue operations, 131 multiple tooth extractions, extraction sockets showing evidence of slow healing, and impacted molars of tissue and bone type.

The preoperative stage is the best time to arrange for a successful operation and a good postoperative result. In addition, we have found that close cooperation with the patient's family physician is of great advantage.

*Allantomide—Prepared by The National Drug Company, Philadelphia, Pa.

In all cases a history was taken, particular stress being placed on the chief complaint, present disorder, and any information relative to past disorders. After the taking of the history the patient was thoroughly examined, starting with a careful examination of all external surfaces of the face, neck, and lips, palpation of the submaxillary areas, then a thorough examination of the mouth starting at the lips and working back to the fauces. In the examination of the mouth the surgeon should have a clear conception of the lesion and should detect every departure from normal. In making a diagnosis, several pertinent points were kept in mind: the local lesions; presence of any systemic disturbance and the possibility that this may be caused by the lesion present; possibility that the lesion is of either general or constitutional origin; and the mode and nature of its local manifestation. The physical examination was supplemented by intraoral x-rays and determination of clotting time, and, if necessary, complete blood work and urine analysis.

Gingivectomies.—Twenty-seven cases of pyorrhea were treated: twelve in routine manner, and fifteen with allantoin-sulfanilamide ointment postoperatively.

The operative field was prepared with proper antiseptics, after which an anesthetic was administered. The operations were performed with an electro-radio surgical knife, being careful not to disturb normal tissue to any great extent. Postoperatively, the gingival tissue slough was removed and interproximal spaces cleaned.

Twelve patients were advised to apply butter, campho-phenique, or glycerin as postoperative measures. Of these patients, three developed complications which were successfully treated with allantoin-sulfanilamide ointment.

In the fifteen patients treated with the ointment, the procedure was as follows: Immediately after the operation, the ointment was applied to the gums with a sterile cotton applicator, and worked into the interproximal spaces. The patients were advised to follow this procedure after meals and before retiring. The comments from this group were that pain was relieved to a great extent by the first application, healing was rapid, and no foul odor was present. No complications developed, and healing time was reduced by at least 40 per cent.

Extraction Sockets.—Multiple extractions because of the presence of advanced pyorrhea, also impacted molars of soft tissue and bone type totaling 131 patients and representing 1,051 extractions were treated.

The patient's mouth was prepared for surgery, then the anesthetic was administered, either local or general, depending on which was indicated. After the anesthetic had taken effect, the gums around the teeth to be extracted were reflected, and the teeth removed. Exodontia sponges or aspiration apparatus were used to keep the operative field dry and free of debris. The extractions were followed by removal of any pathology present, trimming all sharp edges of bone, with excessive gum tissue trimmed and sutured when necessary.

Although the use of sulfonamide powders had been discontinued in extraction sockets, it was decided to try them again as controls for the allantoin-sulfanilamide ointment group. The sulfonamide powders were tried in twenty-five of the patients with a repetition of previous experiences. In three of these patients, the sulfonamide powder "caked," and caused the patients considerable pain which was relieved on removal of the "caked" sulfonamide. There was delayed healing in twelve of the patients which was manifested by dull appearance of tissues. Ten patients had satisfactory results with the powdered sulfonamides.

Allantoin-sulfanilamide ointment in a water-miscible, nongreasy base was used in 106 patients. After the teeth had been removed and the sockets treated postoperatively as outlined above, the sockets were filled with the ointment, using a sterile applicator for this purpose. When the socket had been filled to overflowing, a sterile dry sponge was placed over the sockets, and the patient instructed to bite down on it to produce gentle pressure, which was maintained for fifteen to twenty seconds. The sponge was removed, and the sockets examined to see if clot was formed. This was repeated when necessary. Seldom were more than two applications necessary. The patient was permitted to spit, with the caution not to create suction which would disturb her clot. The excess ointment was gently swabbed away, and, where suturing was indicated, this was done. The operative field was kept free of saliva, blood, and debris by means of aspiration; at no time during the operation was irrigation employed.

Postoperative instructions given the patients included: Precautions not to disturb the wound since doing so may start bleeding. Rinsing of the mouth was prohibited for at least twenty-four hours, after which mouthwashes or warm salt water rinses were permitted four times daily. To prevent swelling, the patient was instructed to apply an ice bag or a towel wrung out of very cold water over the affected part for fifteen minutes every hour if necessary. The patient was requested to return the following day for a postoperative examination. The wound was examined, gently swabbed, and allantoin-sulfanilamide ointment was applied.

In the allantoin-sulfanilamide ointment-treated group that were no complications; pain was readily controlled, healing progressed very satisfactorily, no foul odor was evident in any of the patients, and no dry sockets developed. The healing time in this group was considerably shorter than in the sulfonamide powder-treated group. It is not intended to convey the idea that this therapy is a preventive of dry sockets, but we do feel the incidence of such sockets can be considerably reduced.

Maxillary Sinus Pathology.—Fifteen patients with maxillary sinus pathology of dental origin were treated with the allantoin-sulfanilamide ointment. The sinuses were gently irrigated with normal saline, care being taken not to produce too much pressure. After a complete and satisfactory washing of the sinus, it was packed with a drain which had been "saturated" with the ointment. This was left in place for two days, withdrawn, and the sinus again gently irrigated with normal saline. Following the third or fourth treatment, the washings from the sinus were generally free of debris and the foul odor was absent. Only two patients of the fifteen treated failed to respond to this treatment. These two failures finally responded when more radical treatment was instituted.

Caldwell-Luc Operations.—Seven Caldwell-Luc operations were performed, but instead of petrolatum gauze-iodoform gauze packs, allantoin-sulfanilamide ointment was used to pack the sinuses. The usual intranasal method of irrigation was used to clean out the sinus at two-day intervals. It was observed that pain was considerably relieved; postoperative swelling was not as extensive; the odor was readily controlled; and healing was considerably facilitated by this treatment. Sulfanilamide determinations at four-day intervals revealed less than 1 mg. per cent in the blood.

Compound Fractures and Osteomyelitis.—Similar procedures were followed in the treatment of compound fractures and osteomyelitis. The wounds were treated preoperatively by regular surgical methods. After adequate drainage

had been established, the wounds were irrigated with allantoin 0.4 per cent solution, then packed with the allantoin-sulfanilamide ointment. A rubber dam drain was sutured to the skin. The wounds were irrigated daily with the allantoin 0.4 per cent solution, and packed with the ointment after irrigation, then dressed with sterile gauze dressings. This treatment was continued for two weeks, after which the dressings were changed at three-day intervals until healing was complete. As a supplement to the topical treatment of the wounds, the patients were given orally 1 Gm. sulfadiazine every six hours for the first three days after surgery.

The infection in all four cases was apparently controlled, and within one week the wounds were clean, with evidence of healthy granulation tissues being present. The foul odor so frequently associated with osteomyelitis was controlled, thus adding to the comfort of the patients.

In those patients in whom it was necessary to use liberal amounts of the allantoin-sulfanilamide ointment to obtain complete healing, sulfanilamide determinations of the blood were made at intervals. None of the patients thus studied revealed any appreciable amount of the drug in the blood stream. No toxic reactions, either local or systemic, were observed in any of the patients treated with the allantoin-sulfanilamide ointment.

SUMMARY

Allantoin-sulfanilamide ointment in a special nongreasy, water-miscible base was used as an adjunct to surgery in 184 patients.

The fact is stressed that sound judgment and good surgical practice are of utmost importance, and the use of any drug or combination of drugs is secondary.

Sulfonamide powders or crystals showed a tendency to produce "caking" and to delay healing, although infection was apparently controlled.

The allantoin-sulfanilamide ointment not only controlled infection, but also stimulated healthy granulations, shortening the healing time.

Pain was alleviated in a large percentage of the patients, and the foul odor so frequently encountered in oral surgery was readily controlled.

The sulfanilamide in the blood of those patients tested was negligible. Toxic reactions, either local or general, were not observed in any of the patients treated.

CONCLUSIONS

Allantoin-sulfanilamide ointment in a special nongreasy, water-miscible base, used as an adjunct in oral surgery, controls infection, hastens healing by stimulating healthy granulation tissues, readily controls the foul odor usually encountered in oral surgery, and is easy to apply.

It is suggested that allantoin-sulfanilamide ointment is a worth-while addition to the armamentarium of the oral surgeon for the use in oral surgery.

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1613 S. BROAD STREET

THE PREVENTION OF ENDODONTAL PATHOSIS

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THE treatment of diseases of the pulpal tissues is the phase of endodontia that has received the greatest amount of emphasis inasmuch as common interest has been directed toward learning what should be done about the situation after it has arisen. It is interesting to observe that as simple and elemental as is knowledge of preventing the occurrence of pulp disease, it has received no such similar interest or emphasis.¹ When viewed in the light of the more recent preventive trends in the many chains of the medical sciences of which dentistry is an important one and endodontia an indispensable link, this lack of emphasis on prevention of pulp disease assumes proportions of acuteness and importance demanding immediate attention.

CHARACTERISTICS OF PULP TISSUE

Before discussing the subject of prevention, it is imperative to know the important peculiarities of structure and environment that characterize the pulp tissues.

The connective tissues forming the umbilical cord, the vitreous humor of the eye, and the dental pulp are of an embryonal type.² In the case of the eye and the pulp, these tissues represent a persistence of embryonic tissue in the adult body. Because of the pulp's embryologic characteristics it is to be expected that following the removal of traumatic influences there would probably be a regeneration of the endodontal tissues.

It is a recognized fact that in a large number of cases the pulps of growing teeth recover from injurious influences. However, this is not the case with mature pulps as it is relatively an easy matter for such pulps to succumb following the slightest injury. The connective tissue of the mature pulp tends to

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lose its embryonic characteristics and become fibrous. Such changes together with the inability of the endodontal tissues to respond to inflammatory changes as do other tissues—this because of their encasement in gradually diminishing, hard, unyielding walls of dentine—contribute to the lowering of the powers of resistance and healing of the endodontal tissues.

Combined with these structural characteristics are the following factors³ which contribute to the difficulty of pulpal conservation: (1) the absence of lymphatics within the mature pulp, (2) the absence of a collateral circulation in the arterioles, (3) the impossibility of establishing efficient drainage, and (4) the difficulty of gaining ready access to the diseased organ for the purpose of applying remedial procedures. As a result of these considerations, it may be stated that the prognosis of an acutely or chronically inflamed mature pulp is poor, and its removal absolutely necessary if a state of health of both the tooth and the particular individual concerned are to be preserved.

RESULTS OF REMOVAL OF THE PULP

The removal of the pulp is apt to produce undesirable functional and esthetic changes to the tooth involved. Fortunately, one of these changes, that of coronal discoloration of devitalized teeth, has been successfully overcome in some cases by improved technical methods of bleaching. However, there are other important changes that cannot be as successfully dealt with. It is a clinical observation that pulpless teeth do not have the same powers of resistance to recurring carious processes as do teeth with normal pulps. In the former, there is lost the possibility of secondary dentine formation which is a reflex action responsible for odontoblastic stimulation.⁴ Again, as a result of the loss of dentine vitality, that tissue does not efficiently support the enamel which becomes brittle and therefore easy to fracture. Finally, because of the lowered resistance in the apical region, a pulpless tooth may be regarded as a potential site for the development of periapical disturbances.

It is to be wondered why a more intensive campaign for methods of preventing pulp diseases has not been consistently carried out when it is realized how relatively difficult an operative procedure it is to remove a tissue as delicate as the pulp, and the associated disadvantages following its removal.

ETIOLOGY OF ENDODONTAL DISEASES

In addition to the above facts relative to the structural characteristics of the pulp tissues, an accurate knowledge of the etiological factors of endodontal pathosis is another prerequisite to the formulation of any plans for a program of prevention.

These factors may be conveniently divided into six major groups: (1) Dental caries. (2) Thermal shock. (3) Electrical irritation. (4) Trauma. (5) Bacterial invasion. (6) Chemical irritation.

1. *Dental Caries.*—The most common cause of inflammation of the pulp is dental caries which has progressed sufficiently to involve the organ or to expose it.⁴

Dental caries has been defined as a local disease of the teeth in which the enamel is dissolved by lactic and pyruvic acids, as waste products of microorganisms and the dentine is disintegrated by the vital activity of proteolytic organisms penetrating the dentinal tubules.⁵ As a result of a carious process extending to involve the pulp, the latter is invaded not only by the microorgan-

isms directly responsible for acidogenesis with accompanying calcific disintegration, but also by the many other types of organisms found in the normal flora of the mouth.

The extensive work carried out by the Michigan Dental Caries Research Group has fully demonstrated that the quantitative estimations of *Lactobacillus acidophilus* is a relatively accurate criterion of the degree of activity of caries in the mouth.⁶ Prominent among the conditions which influence the activity of acidogenic bacteria is the factor of the carbohydrate food supply. It is a well-known fact that *L. acidophilus* thrives best on media containing carbohydrates, especially the simple, easily fermentable sugars, therefore the amount of available fermentable carbohydrate must be considered in estimating the factors favoring the growth of this organism.

Although there is not complete agreement that *L. acidophilus* is the specific cause of dental caries, there has been established a relationship between the disease and the presence of this organism in the mouth. From the standpoint of prevention it would be logical to state that a lesser carbohydrate intake contributes to the lesser number of these organisms, and this in turn is a factor that is important in controlling dental caries. If the disease can be controlled in this way, then there is the definite possibility of reducing the number of pulp exposures from dental caries.

Dental caries is responsible for another abnormal condition of the pulp, namely, hyperemia. This condition results from the stimulation of the sensitive fibrils of the odontoblastic cells of the pulp by the acids of the carious process. A pain reaction is the usual symptom.

A thorough oral examination in which bitewing roentgenograms are included is necessary from a prophylactic standpoint, to detect incipient carious processes so that corrective procedures may be instituted before undesirable symptoms develop.

2. *Thermal Shock*.—Pulp irritation as a result of thermal changes is an extremely common observation. It is normal for extremes in temperature changes to produce pain reactions in the pulpal tissue. It is when these reactions are forced beyond the limit of normalcy that the pathologic changes produced in the pulp reach a stage of irreparability and some form of radical therapy must be resorted to.

Various careless dental operations are also responsible for a large number of the cases of pulp irritation resulting from thermal changes. Dr. Paul Jeserich⁶ has done a great deal of work on the thermal changes occurring within the pulp as a result of frictional heat produced by the use of burs and stones. Greater care and judiciousness in operative work by dentists can reduce these ill effects on the pulp. Another important observation is the lack of consideration given to deep cavities. I have observed a large number of amalgam and inlay restorations in rather deep cavities in which no cement bases were used, and the entire cavity was completely filled with the metallic filling. Invariably, the patients complained of extreme sensitiveness in those teeth long after the restorations were placed. The conduction of temperature changes to the pulp by the metallic filling material was possibly responsible for the pain symptoms.

From the standpoint of prevention, it may be stated that patients should be carefully instructed in the deleterious effects produced on dental pulps as a result of the subjection of the teeth to extreme changes in temperature. Dentists must be more greatly impressed with the profound responsibility of dental operators to patients and carefully effect their operative procedures, constantly

keeping in mind the anatomic and physiologic considerations which form the biologic background for successful technical dental operations.

3. *Electrical Irritation*.—The affecting of the dental pulp by a static form of electrical impulse may be illustrated by the sharp pain produced in a tooth with a metallic filling on the application of any metallic dental instrument to the filling. A similar effect is produced by the contacting of dissimilar fillings in opposing teeth. The difference in electrical potential between the two metals when brought into contact produces a charge of static electricity which in turn produces a pain reaction in the pulp.⁷ A galvanic form of electrical impulse is the stimulus produced on the pulp as a result of two dissimilar fillings in adjacent teeth. Chronic irritation may be produced in the pulps of teeth as a result of the generating of a weak current between the two fillings, the saliva acting as the electrolytic fluid.

These are both cases where prolonged irritation of the pulp by this form of irritant can be prevented by judicious treatment planning. A careful oral examination and subsequent restorative work after thoroughly considering every factor that might in the slightest way influence a scientific plan of treatment will accomplish this purpose.

4. *Trauma*.—Under trauma is included injury produced on the endodontal tissues by accidents, abuse of the teeth, and abnormal chewing habits. Of frequent occurrence are accidents which result in the fracture of teeth and exposure of the pulpal tissues. It is a relatively easy matter for a fall from some height or a direct blow on a tooth to produce a break of the enamel or dentine or both. In addition to this, similar accidents which do not break the tooth structure might sever the connection of the various structures at the apex. This, in turn, results in the complete cessation of nutrition to the pulp and the latter's death.

These effects on the endodontal tissues may be similarly produced in individuals who use the teeth for purposes such as chewing bones and hard candies, cracking nutshells, and removing caps from bottles. Certainly these hard tissues were not constructed for such functions. The third factor is abnormal chewing habits. If anterior teeth are used for chewing, or if any one tooth is subjected to an occlusal trauma, this resulting from abnormal chewing habits, there is a distinct possibility that some effect may be produced on the pulp as a result of injury to the periapical vessels and nerves.

The information given patients relative to extreme caution in the use of the teeth and the avoidance of accidents and injurious masticatory habits is the only way that the preventive angle of pulp disease resulting from these factors can be stressed.

5. *Bacterial Invasion*.—The invasion of the pulp by the organisms of a carious process, and by other normally occurring oral organisms after the carious process has widely exposed the pulp, has already been discussed. Mention should be made of the great possibility of infective invasions of the dental pulp via the foramina of the root apices from adjacent infective processes and from other distant foci by way of the general circulation. Henrici and Hartzell⁹ found that the pulps of normal teeth were free from infection, but, in the pulps of nonexposed carious teeth with periodontoclasia, bacteria could be recovered from the pulps in a large number of cases. This did not necessarily mean that there was infection in any or all of these cases.

The possibility of the pulp being affected by systemic conditions may very conveniently be included here. Lowering of the already poor resistance of the

tissue may result from the blood dyscrasias, systemic poisons, age, menstruation, various febrile conditions, streptococcal infections, and other abnormal conditions.

From the standpoint of prevention, little can be said for these cases, as endodontal disturbances resulting from these conditions are not common and, when present, would be difficult to discover without having to resort to an elaborate dental and physical examination.

6. *Chemical Irritation.*—Irritation of the pulp from dental caries, which is a chemicoparasitic process, has already been discussed. However, there are other forms of chemical irritation from which the pulp may suffer. When the dentine of the tooth becomes exposed, either from caries, erosion, or abrasion, it becomes hypersensitive and will give violent pain reactions to salt, sugar, the acids in cement bases, the saliva, and various caustic or irritating drugs. Misuse of the toothbrush may cause abrasion of the enamel and an exposure of the dentine. From a preventive standpoint, it may be stated that individuals should be taught the correct manner of toothbrushing and warned of the danger of misuse. Since the etiological factors of erosion and abrasion are not definitely known, very little can be said at this time about preventing their occurrence, this in turn rendering less likely the exposure of the dentine to irritating chemical influences.

In addition to the prevention of these factors that do cause endodontal pathosis, there are *two* conditions that should be stressed in any program that has for its function the prevention of pulp disease. One of these factors is the maintenance of a good pulpal circulation. This may be accomplished indirectly by frequent massaging of the gums. The close relationship existing between the blood supply of the alveolar bone, the gums, the periodontal membrane, and the pulp makes it logical to assume that massage of the gums will give some little assistance to the renewal of the circulation to the pulp. In addition, the pressure and relaxation forces exerted on the periapical tissues and vessels during active mastication aid in forcing in new blood and letting out deoxygenated blood from these parts. In this manner, stagnation of blood is prevented and the maintenance of high tissue tone maintained.

The other factor referred to is that of frequent and adequate dental examinations. At the present time, the importance of frequent dental examinations, although realized by the dentist, is not yet fully understood by the public. The use of bitewing roentgenograms, transillumination, pulp testing apparatus, and sharp explorers should form the basis of an adequate dental examination, yet many dentists are reluctant to carry out these necessary procedures. Consequently, it appears that the dentist needs to be more fully convinced of his responsibilities, a knowledge of which is a prerequisite to the successful performance of his duty of instruction to the public so that they may realize the importance of his functions.

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CANCER OF THE ORAL MUCOSA AND CIRCUMORAL AREAS

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CANCER is a disease that is perhaps more dreaded by more people than any other ailment. Next to the recent war casualties, cancer has the highest death rate and ranks second only to heart disease as the Nation's No. 1 killer. More than 165,000 American men, women, and children die annually from this disease. One person dies of cancer *every three minutes!* Medical authorities believe that about 100,000 die needlessly, as most of these deaths could have been prevented and the patients cured by prompt diagnosis and skilful treatment.

According to Pfahler,¹⁵ about 4,000 people in the United States die each year from cancer of the lip and mouth.

"Such symptoms as localized pain, a loose tooth, abscess, and swelling of the lower jaw, in the vast majority of cases may have nothing to do with cancer, but one must think of cancer and must institute those diagnostic surveys that will detect its presence—roentgen-ray films of the teeth and of the upper and lower jaws; coverslip examinations of the mouth, not only for Vincent's infection, but also for the pyogenic organisms, blood tests for syphilis, blood counts for the presence of anemia and leucemia, and studies to determine the possibility of scurvy. An oral survey must be as comprehensive as a medical survey. According to our present-day conception of thorough physical examination, one is incomplete without the other. I believe, on the evidence available today, the control of cancer of the mouth is the problem of the dental profession, because the dentist is the first to see those enlightened persons who come early for treatment."¹¹

Dr. James Ewing has said: "The responsibility for detecting oral and intra-oral cancer falls chiefly on the dental profession."¹⁰

In my opinion, not only the dentist but also the physician and the oral surgeon must become cancer conscious and able to recognize the gross appearance of cancer of the oral cavity.

Early diagnosis of malignant lesions is an important deciding factor in determining the seriousness and fatality by which the majority of these cases terminate (Fig. 1).

Cancer, in general, occupies second place in the death rate in the United

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States. Fear, delay, and ignorance, as always, continue to be the allies of disease and the enemies of cure. Cancer is the "fifth columnist" of disease. It is not contagious; it is not hereditary; it is not caused by a microorganism. The United States report on Vital Statistics for 1939 indicated that over 5,012 deaths were due to intraoral cancer. In accordance with these facts, the dental surgeon plays an important role in the early detection of cancer and thus in its prevention.



Fig. 1.—Early squamous-cell carcinoma with no metastasis in a male patient 53 years old (referred by Dr. Leslie Boone). To avoid seriousness and fatality by which the majority of oral cancers terminate, early detection (such as in this case) and prompt treatment are the only solution. The lesion was surgically removed without recurrence.



Fig. 2.—Metastasis. Showing regional glandular involvement of the submaxillary lymph nodes in a patient 52 years old (referred by Dr. R. Rochester). This patient had a malignant lesion in the floor of the mouth for several years.

A tumor or neoplasm may be defined as a disorderly growth of new cells which proliferate without control and which serve no useful function. The word "cancer" is used to include all the various types of malignant growths occurring in any part of the body, resulting from certain changes that the body cells undergo. This paper shall deal particularly with cancer in and around the oral cavity and its immediate spread to the face and neck.

These changed cells multiply and destroy neighboring healthy cells and

keep growing. Some grow rapidly, some slowly, some spread (metastasize) quickly through the body (Fig. 2); others do not (Fig. 7).

The English word "cancer" is from the Latin word *cancer* meaning "crab" and these extensions are the claws of the animal. If the cancerous lesion is left unnoticed, it will grow and spread to remote parts of the body by means of the following methods:

1. By the lymph (lymphogenous).
2. By the blood (hematogenous).
3. By continuity, cells growing from one tissue to another.
4. By lymphatic embolism—the tumor cells are carried to the regional lymph nodes and sometimes to more distant nodes.

This spread is called metastasis.



Fig. 3.



Fig. 4.

Fig. 3.—Cancer of the lip is the most common form. It may also appear in other parts of the oral cavity, such as the tongue, cheek, alveolar processes, soft and hard palate, and floor of the mouth. This lip lesion was treated as a chancre previously by mistake.

Fig. 4.—Papillary carcinoma of the cheek and retromolar region in a patient 51 years of age. Local recurrence after cure of a leucoplakia area. Patient gave a history of excessive cigar smoking for over twenty years.

OCCURRENCE

No age is free from cancer (Fig. 6). Babies are born with it and the oldest people die with it. However, it is a disease of adult life, occurring chiefly between the ages of 40 and 70 years. Oral cancer is more predominating in men and rarely seen among women. Cancer of the lip is the most common form (Fig. 3). Other parts of the oral cavity where cancer occurs are the tongue, labial and buccal regions (Fig. 4), floor of the mouth, the hard and soft palate (Fig. 5), the larynx, and the pharynx.

ETIOLOGY

Exact etiology of cancer is not known. However, the following have been considered to be associated by many observers as possible causes to a large majority of intraoral cancer:

1. Thermal, chemical, or mechanical irritation from excessive use of cigarettes, cigars, and pipe smoking.
2. Irritation from decayed, jagged, protruding, or broken teeth, and the wearing of loose and ill-fitting artificial dental restorations.

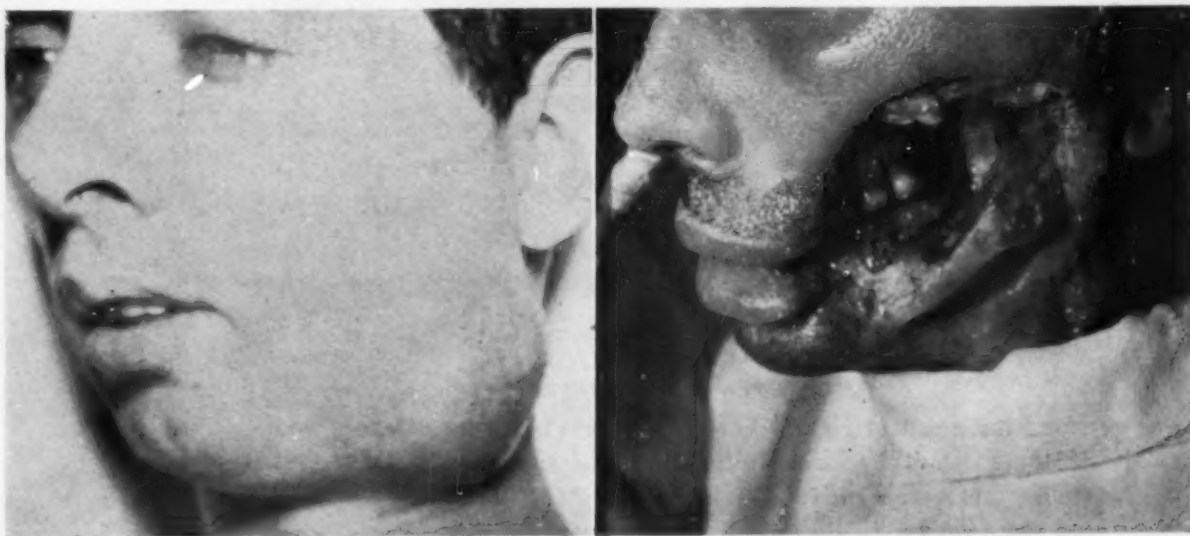
3. Precancerous lesions are examples of chronic irritation such as ulcers, cracks, leucoplakia, and particularly syphilis, all of which are constant antecedents.
4. Objects held in the mouth in certain occupations, such as pins, nails, or sulfur.
5. Uncleanliness and malhygiene of the mouth.
6. Chronic sepsis and oral sepsis of the gums and teeth.



A.

B.

Fig. 5.—*A*, Carcinoma of the maxillary sinus in a female patient 36 years of age. *B*, This picture was taken only four months later and shows rapid and extensive invasion of the lesion through the cheek, hard and soft palate, nasal fossa, and orbit. The patient died six months later.



A.

B.

Fig. 6.—No age is free from cancer. *A* shows a boy 8 years of age with a large lymphosarcoma involving the submaxillary lymph nodes. *B*, A squamous-cell carcinoma of the lip in a patient 58 years of age. Note extensive invasion to surrounding structures. The patient died one year later. Early detection of the lesion and immediate treatment could have saved this patient.

DIAGNOSIS

Not all oral lesions are malignant. For this reason, malignant oral lesions must be differentiated from ulcers, tuberculosis, syphilis, or benign growths.

If prophylactic or therapeutic measures fail to clear up a lesion, biopsy must be resorted to for exact diagnosis.²

Early malignant oral symptoms are mild, and as there is rarely any pain at the time, they are frequently mistaken for other harmless ailments.

Detection of early cancer is often difficult, especially in some cases where early symptoms are not present.

The gross appearance first presents a local induration, then a warty mass followed by deep infiltration. The malignant ulcer is slow-growing, hard, indurated, invasive with round edges. It may and may not involve the lymphatic nodes. Extensive necrosis, sloughing, destruction, and secondary infection with metastasis follow. The malignant ulcer may destroy the lip, the skin of the chin, the cheek, and finally involve the maxillary sinuses (Fig. 5), nose, eye (Fig. 15) and mandible (Fig. 6, *B*).

The submental and submaxillary lymph nodes are involved, depending on the location of the malignant lesion. The following are good diagnostic features for possible malignancy.

1. Any sore about the tongue, mouth, or lips that does not heal in ten days to two weeks.

2. Difficulty in swallowing or hoarseness that has developed without any apparent cold or infection.

These are unnatural conditions. If detected early and properly diagnosed, cancer is often cured or at least controlled.

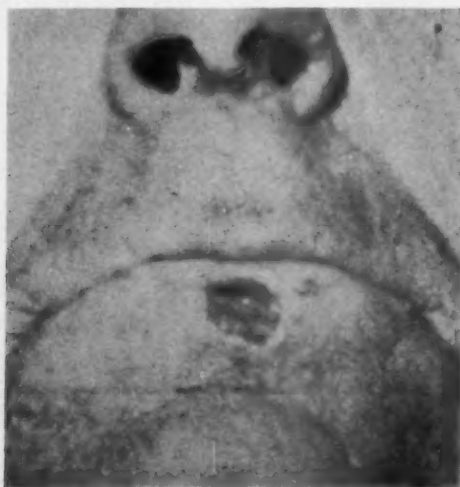


Fig. 7.—A squamous-cell carcinoma of the lower lip with no metastasis in a male patient 53 years of age (referred by Dr. George Paschall).

PROGNOSIS AND PREVENTION

Although malignancy of certain parts of the oral cavity is serious, it is one of the most easily preventable forms of cancer. The prognosis, in the early states, therefore, is favorable. In spite of the fact that oral cancer and its causes are well known to the medical and dental professions, and that the tissues of the mouth are accessible for early detection of any abnormal condition, intra-oral cancer continues to present an important problem.

The most satisfactory results are obtained when the lesion is detected early and treated promptly. A sound and suggestive approach to the problem of cancer control depends on two groups, as follows:

1. That the medical and dental professions be made more cancer conscious and able to recognize not only the earliest symptoms but also the gross appearance of malignant lesions and their treatment.

2. That the public be taught the importance of the early signs of malignant disease and that "early cancer is curable."

The failure of the patient, therefore, to consult a physician, who in turn sometimes neglects to make a necessary examination, gives an unfortunate result.



Fig. 8.



Fig. 9.

Fig. 8.—V-shaped excision of the lesion of the lower lip of case in Fig. 7. This was followed by radium treatment. The excision was done under a local anesthetic. The biopsy revealed the lesion to be a squamous-cell carcinoma.

Fig. 9.—The coronary artery of the lip was tied with fine catgut. From the inside, deep approximation of the tissues was obtained by catgut sutures. On the skin, fine silk sutures were used and careful approximation of the parts was obtained.



Fig. 10.



Fig. 11.

Fig. 10.—The skin sutures were removed after six days. A vertical hairline scar in the lip is hardly noticeable one month after operation.

Fig. 11.—Five years after the proper plastic repair of the lip was done. There is no sign of scar or evidence of recurrence.

CLASSIFICATION AND GRADING

Tumors are divided into two classes; the one, innocent, simple, or benign; the other, malignant. This is true for epithelial and connective-tissue tumors. A tumor may show any degree of malignancy, which in turn has an important bearing on the prognosis and on the prospect of successful treatment.

For this reason Broders⁸ has introduced a system of grading malignant tumors, dividing it into four groups or grades according to their apparent malignancy. Grade 4 is the most malignant; grade 1, the least. Grading is a useful method of communication between pathologist and surgeon and is also

used as a guide to treatment. Grades 1 and 2 being suitable for operative removal, and grades 3 and 4 for radiation.

Boyd⁹ groups the tumors of the oral cavity from the point of view of malignancy as follows:

1, Tumors from lips to teeth—mostly low grade; 2, tumors from teeth to back of tongue—increasing in malignancy as we pass back; 3, tumors of pharynx—high-grade malignancy.

Tumors are either benign or malignant and originate from the epithelial or connective tissue cells.



Fig. 12.—Basal-cell carcinoma (rodent ulcer) in a patient 58 years of age. This lesion appears to be of slow growth and has the characteristic rolled border. It usually appears on the upper part of the face about the cheek, nose, eyelid, and ear. (Courtesy of Dr. R. Larer.)



Fig. 13.—Melanosaarcoma of the gingival tissue in a patient 39 years of age. Note bluish-black pigment and nodular appearance. This is generally recognized as the most malignant of all sarcomas.

EPITHELIAL TUMORS

Among the most important tumors derived from epithelial cells are the papilloma, the adenoma, and the carcinoma.

A papilloma is an innocent or benign tumor and may be squamous or mucous depending on whether it grows from a squamous or a mucous surface. A papilloma, however, may become malignant.

A squamous papilloma is most common in the skin but may occur in the mouth, larynx, or any other cavity lined by stratified epithelium.

A mucous papilloma is one that grows from any mucous membrane.

An adenoma is an innocent epithelial tumor of glandular structure.

The carcinoma is a malignant epithelial tumor (Fig. 1). It is the commonest of all malignant tumors; much more so than sarcoma (Fig. 6, A). Among the most important carcinomas, are the squamous-cell and basal-cell carcinomas.

Squamous-Cell Carcinoma.—This is called epithelioma and epidermoid carcinoma. It grows particularly on the skin, lip, tongue, larynx, cervix, and urinary bladder (Fig. 3).

The Basal-Cell Carcinoma.—This is also called "rodent ulcer." It is of slow growth. The lesion occurs on the upper part of the face about the cheek, nose, eyelid, and ear, above a line drawn between the tip of the ear and the angle of the mouth (Fig. 12).



Fig. 14.—Early carcinoma of the lower lip before and after treatment with diathermy, x-ray, and irradiation. There is no evidence of metastasis. Complete cure resulted without recurrence. (Courtesy of Dr. Frank Bernard, Chicago, Illinois.)

CONNECTIVE TISSUE TUMORS

Tumors derived from connective tissue cells are the osteoma, fibroma, angioma, lymphoma, sarcoma, and others. All are benign except the sarcoma.

Most sarcomas are mixed growths. The character of the cells predominating determine the degree of malignancy. The melanosarcoma is considered one of the most malignant of all sarcomas (Fig. 13). There are various varieties in order of their malignancy. These are as follows:

1. Giant cell.
2. Spindle cell.
3. Round cell.
4. Mixed cell.
5. Alveolar.
6. Melanotic.

TREATMENT

Thousands of men and women have been treated successfully for cancer.

The following are the methods used in the treatment of cancer:

1. Surgery—surgical removal of the lesion.
2. Radium—destruction of the lesion.
3. X-ray—treatment of the lesion.

In its early stages it is usually limited to one spot, and can, therefore, be

removal or destroyed by surgery, x-ray or radium alone, or in combination (Fig. 16).

Table I shows the percentage of cure in early and late treatment of oral malignancy.

TABLE I

| TYPE | EARLY TREATMENT PER CENT | LATE TREATMENT PER CENT |
|--------|-----------------------------|----------------------------|
| Mouth | 80 | 20 |
| Lip | 85 | 10 |
| Tongue | 80 | 10 |



Fig. 15.—An attempt in removing a malignant cheek lesion in a patient 31 years of age by means of surgery and radium treatment. It resulted in the destruction of the eyelids and entire orbital contents. The lesion was first noticed five years previously.

Methods of Treatment.—

1. Mouth Hygiene.

- (1) Establishment of oral hygiene.
- (2) Removal of all irritating teeth and artificial restorations.
- (3) Removal of all pathology present.

2. Local Treatment of the Lesion.

- (1) Surgery—surgical removal of the lesion (Figs. 7 to 11 inclusive).

This is advisable in early malignancy of small lesions and where there is no metastasis present. Involvement of the lymph nodes is always serious. In unilateral, freely movable, secondary lymph nodes of the neck, and where the prognosis is favorable, the operative removal of the lymph node is indicated.

In contraindicated cases, irradiation by means of x-ray or radium is the method of choice as shown in Fig. 14.

(2) Irradiation.

(a) X-ray therapy.

The lesion may be treated by means of x-ray by the following methods (Fig. 14) (the dosage is determined by the site and extent of the lesion and the method employed):

Direct contact low voltage x-ray.

Intraoral high voltage x-ray.

High voltage x-ray applied externally at different sites of the neck, the rays being directed through the lesion.

(b) Radium therapy.

Where radium therapy is the method of choice, it is applied as follows (Fig. 11):

Interstitial application.

Direct application of surface applicators to local lesion.

Placing of applicators to outside of neck.

Whenever possible, the treatment of cancer should begin in the precancerous stage, that is, where there are sufficient symptoms which might lead to malignancy.

There have been remarkable cures by the use of radium (Fig. 16), and also many disastrous failures (Fig. 15).



Fig. 16.—Arrested squamous-cell carcinoma of the maxilla. The lesion was surgically removed and followed by radium therapy. The patient is well at present, eleven years after operation without recurrence. He did not consent to further plastic facial reconstruction. (Courtesy of Dr. J. E. Scheafer, Chicago, Illinois.)

The following recommendations reported by Ewing¹⁰ seem to be along conservative lines, as follows:

1. For early localized cases—radium.
2. Established or borderline cases—operation.
3. Inoperable cases—radium.
4. Advanced cases—neither radium nor operation.

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1930 CHESTNUT STREET

A CLEFT PALATE PROSTHESIS

TREATMENT OF A CASE WITH A HOLLOW-BALL OBTURATOR

BEN B. CANTOR, B.Sc., D.D.S., WINNIPEG, CANADA

THE many methods available for cleft palate prosthesis attest to the usual unsuitability of these on the score either of function, anatomic effect, esthetic result, or voice resonance. Many of the procedures proposed are technically difficult and some are mechanically unsound. The present paper deals with the construction of a hollow-ball metal obturator and illustrates that by this means it is possible to construct an artificial palate which provides complete anatomic and functional restitution.

The construction of a suitable appliance in any given case requires a thorough knowledge of the anatomy and physiology of the palate with especial reference to the missing parts and a clear understanding of the principles governing the construction and operation of obturators. The hard palate is a rigid, bony structure covered with soft tissue. It forms the floor of the nasal cavity and the roof of the oral cavity. The domed shape of the oral surface modifies speech tone giving resonance to the voice. The soft palate is a pliable structure. It includes the uvula and a curtainlike layer of muscular tissue which extends from the posterior border of the hard palate backward and downward to join the sides of the pharynx where it forms the pillars of the fauces. The soft palate thus hangs in the junction of the oral and nasal cavities. In the act of deglutition it prevents food from passing into the posterior part of the nasal cavity. During speech it closes off the nasal passages preventing nasality of voice, or "twang."

The soft palate is essential for the production of normal articulate sounds. During speech the posterior wall of the pharynx rises and moves forward, forming a rounded ridge which meets the soft palate, the latter being drawn upward and backward to make contact. This closes off the nasal cavity and forces the air column through the oral cavity only and gives the speech a characteristic, normal tone. In swallowing, the soft palate forms a low, tense arch, meeting the upward-drawn posterior part of the tongue. The oral cavity is thus closed and the nasal passage is left free. It is thus possible to retain

food or liquid in the mouth while continuing to breathe through the nose. It is thus clear that in instances of cleft palate the individual is deprived of the ability to cut off communication between the oral and nasal cavities. This produces interference in the act of deglutition and creates a speech defect in which the outstanding characteristics are nasality and poor enunciation.

The essential requirements of a good obturator are that it should effectively stop the gap between the defective parts of the hard and soft palates and be long enough so that the posterior wall of the pharynx may come in contact with it. Rigidity and fixation are obtained by casting it together with the denture. In repose, the obturator need not fill the posterior section of the gap, but in speech the muscles of the soft palate cause the separate sections of the cleft to come in contact with the ball-shaped portion of the appliance and slide along its sides. These should be sufficiently flared to prevent the edges of the cleft from slipping off. In this manner, with the pharynx apposed on the posterior end of the obturator and the edges of the defective palate clasping its sides, the patient can occlude the posterior nares. Thus, the most essential requisite for speech restoration is effected. The obturator then acts as a "crutch" to enable the defective parts to function.

CASE REPORT

The patient was a young woman of 23 years, with congenital cleft palate. Surgical closure was attempted on four occasions, between the ages of 2 and 7 years. As a result, the middle third of the cleft was bridged by a strand of mucosa. Apart from this, the cleft extended from a position in line with the second premolars backward into the nasopharyngeal space. The vomer was missing and the arch of the jaw was distorted. There were ten teeth present in the upper jaw. The right second and third molars, the left second premolar and second molar were placed off the alveolar ridge. The right and left cuspids were sound and in occlusion with the lower dentition. The central and lateral incisors were bunched, and protruded over the lower lip. The mouth and teeth were clean.

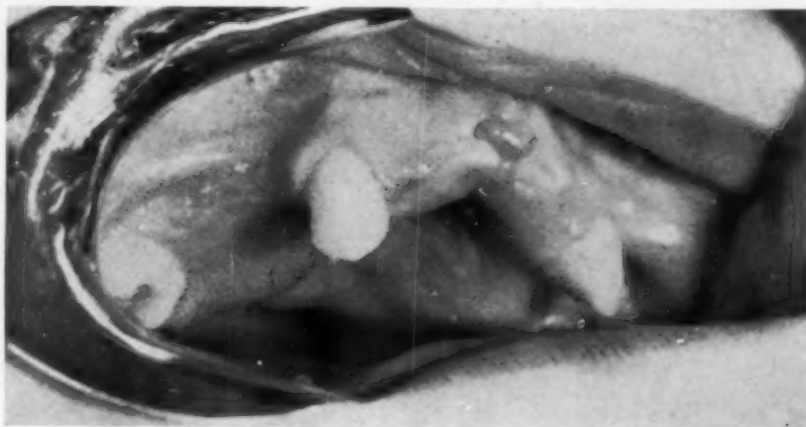


Fig. 1.

Despite the hazard of a possible maxillary fracture it was felt that a better esthetic effect could be obtained if the central and lateral incisors were removed and an alveolectomy performed. This was accomplished without undue incident, and healing was normal. Fig. 1 illustrates the appearance of the upper jaw and palate when healing was complete.

It would have been impossible to secure an accurate reproduction of the vault and dental arch in one impression even with the best-developed colloidal impression materials. Such an impression in any case represents the borders of the cleft in a contracted position because contact with the impression mate-

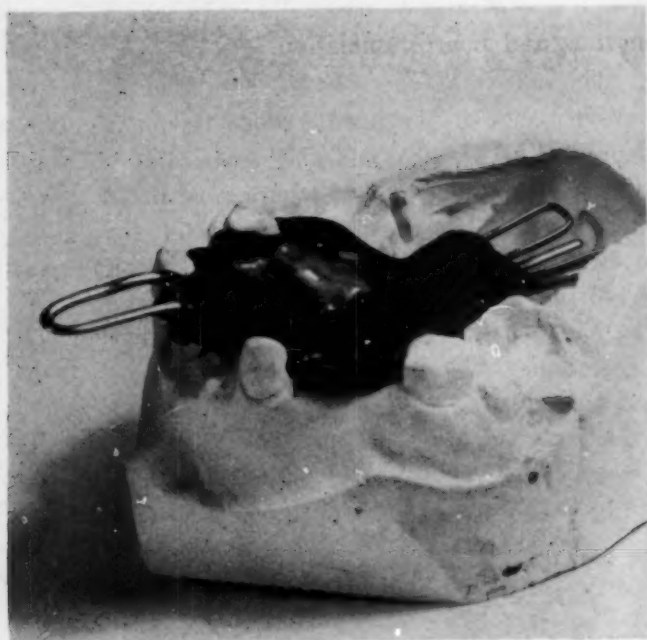


Fig. 2.

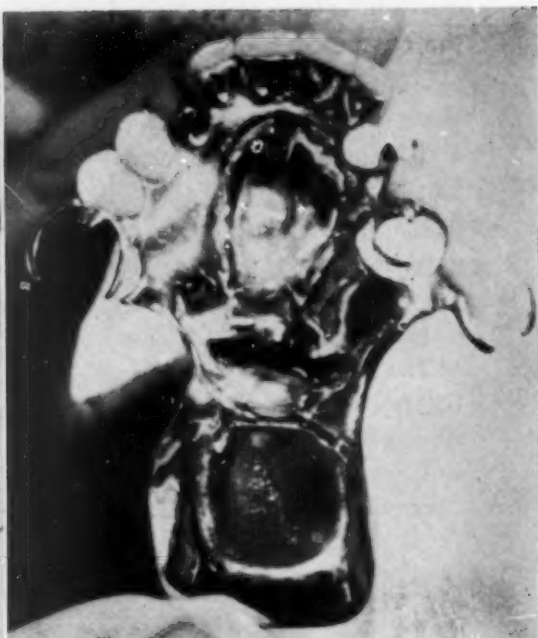


Fig. 3.



Fig. 4.



Fig. 5.

rial causes the soft palate to be drawn upward and backward so that the cleft is partially closed. In addition, the impression, by pressing the tissues back, increases this effect. For these reasons it was felt that the cast should represent the borders of the cleft in their relaxed position and the posterior wall of the pharynx in its contracted state. The primary impression was taken with a black tray-forming compound without regard for the cleft. An acrylic tray was then devised (Fig. 2) with a loop handle in the anterior region and a double loop of stainless bar wire projecting from the posterior part of the tray. Both the handle and the projections were embedded in the acrylic tray in one continuous loop. With this tray, and using a low-heat compound, an impression of the cleft and the vault was obtained in fine detail. Softened carding wax was added to obtain a seal as far back as possible in order to separate the nasal passage from the oral cavity. Finally, a plaster wash impression was taken to include the pharyngeal surface of the soft tissues bordering the cleft. The core of compound on the posterior loop extensions was used for carrying the fine plaster wash to this area and to hold it there while setting, thus minimizing the amount of plaster and controlling its flow.

The impression so obtained was poured in stone. The case was waxed up in the usual manner, taking into consideration the teeth to be clasped and those on which onlays were to be added. Vitallium was selected for the construction of the appliance. Vulcanite or acrylic could not be used because of the processing difficulties in making the posterior section a hollow ball. Gold or platinum alloys might have been used, but vitallium was selected because it combines strength, light weight, thinness, and cleanliness. Effective retention and stabilization of the obturator were obtained by Aker-type clasps, together with distal stabilizers. Clasps, base, and the upper half of the hollow ball were made in a one-piece casting. A lid was then waxed up for the lower half of the ball and this was welded on to complete the hollow-ball obturator.

The completed appliance is shown in Fig. 3. Making the bulb hollow lightens it considerably and improves the tone quality of the speech. Figs. 4 and 5 show the appliance in position.

In use, this obturator proved to be stationary in position, but was so formed that the pharyngeal muscles, in movements incidental to the production of articulate sound, hug the obturator and so separate the cavity of the nose from the oral cavity. After two adjustments the patient accepted the appliance as completely comfortable. After a short period of speech training from a teacher of deaf-mutes, the patient was pronounced cured. Re-examination after six months showed complete anatomic and physiologic restitution.

SUMMARY

1. The anatomy and physiology of cleft palate are reviewed briefly.
2. The objects aimed at in the construction of an obturator prosthesis are outlined.
3. A case of cleft palate is reported. This was treated successfully by the construction of a hollow-ball vitallium obturator.

Case Reports

CASE NO. 94

COMPLETE LUXATION OF THE MAXILLARY ANTERIOR TEETH

CAPTAIN MONTAGUE A. CASHMAN, D.C.

History.—The patient had driven his weapons carrier into the tail of an airplane being towed by a tug, having been blinded by the light of the tug and unable to see the airplane behind it. The airfield was darkened and the blow produced unconsciousness. The patient had been treated at his local sick bay, where sutures were placed on the outside of the chin, prior to which an undetermined "sulfa" powder was sprinkled into the wound. The patient was sedated, and for five days the swelling of the chin and lips increased, as did also the pain. On the sixth day, he was referred to the oral surgery section of this hospital. The following injuries were noted upon admission:

1. Severe lacerating wounds of the chin.
 2. Lacerating wound of the lower lip.
 3. Lacerating wound of the left forearm, right index finger, right knee and chest.
- Pus was present in all of the wounds.

The anterior maxillary alveolar process was greatly denuded, due to loss of soft tissue, and had been exposed to fluids of the mouth. Much fragmentation of bone could be palpated under the remaining soft tissue, high under the mucobuccal fold, and a solid mass was present and clearly palpable under the external cheek, about $\frac{1}{2}$ inch to the left of left nares. The lower anterior teeth and left first and second premolars were very motile, and this area of alveolar process could be felt to move as one unit, labially and lingually. The lower lip, from the midline to the left angle, was edematous and drooped over the teeth beneath, beginning about $\frac{1}{4}$ inch below the vermilion border. The chin was lacerated and presented a through-and-through wound, opening on the inside of the mouth, about 1 inch in length.

X-rays.—

Skull: Negative.

Maxilla: Anteroposterior, occlusal, and intraoral x-rays were taken. The nose and malar bones were negative. There was no horizontal fracture of the maxilla. The left central incisor, left lateral incisor, and left cuspid were driven for their full length into the cancellous portion of the maxilla.

Mandible: X ray disclosed a vertical fracture without displacement, extending from the alveolar process between the left central and lateral incisors, to a point down to approximately the center of the horizontal ramus.

Treatment.—In view of the fact that there was no record of tetanus toxoid having been administered, 1 c.c. of this was immediately given after the initial examination here. Also, as a precaution against gas gangrene, a prophylactic dose (1 c.c.) of trivalent gas gangrene antitoxin was given following the usual skin test. One hundred thousand units of penicillin was administered intravenously in 1,000 c.c. of saline solution. Penicillin was also applied locally and 10,000 units intravenously were given every two hours. Sulfadiazine, 40 grains immediately, and 1 Gm. every four hours, was ordered. Blood and urine studies remained within normal limits. Continuous hot saline soaks were applied to the chin. Continuous hot saline mouth irrigations were given by means of an elevated 1,000-c.c. bottle and rubber tubing extending down into the patient's mouth.

The sutures on the outside of the chin were removed to allow for adequate drainage. A light protective bandage was placed over this area. Three days following admission, sufficient edema had disappeared to warrant operative procedure.

Operation.—Under nitrous oxide-ether administered endotracheally, the mucoperiosteum over the involved maxillary teeth was incised and flapped back. Large areas of fragmented bone were uncovered. The alveolar ridge was reduced to allow the remaining mucoperiosteum to cover the new level of bone. The larger fragments still attached to the periosteum were left intact. Sulfathiazole powder was sprinkled into the exposed areas, and then a continuous silk suture was placed. Healing was uneventful and the sutures were removed on the third day, when a labial arch bar was fitted and the loosened lower teeth were wired to this bar, thus stabilizing the teeth and the fractured segment.

New x-rays were taken which showed position to be good. The patient was returned to light duty one week after placement of the arch bar.

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LABIO-LINGUAL TECHNIC

By OREN A. OLIVER, RUSSELL E. IRISH, CLAUDE R. WOOD
450 Pages. — 278 Illustrations. — Price, \$10.00

This new book defines and describes under the heading of "Labio-Lingual Technic," the use of the labial and lingual appliances in the treatment of malocclusions. The authors have put into concrete form a technic for the treatment of malocclusions that is sufficiently comprehensive to permit a step-by-step description of the introductory phases, construction, and use of the labial and lingual appliances.

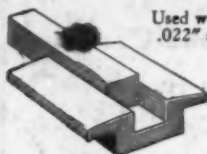
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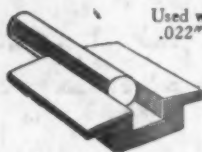
BRACKETS NOS. M 464 and M 465

A modification of the Dr. E. H. Angle Edgewise Arch Bracket for use with other types of arches, made in response to requests for a bracket of this type and after a suggestion by Dr. A. F. Jackson. These brackets are about half the height (consequently project less) and are considerably longer than the Edgewise Arch Bracket.

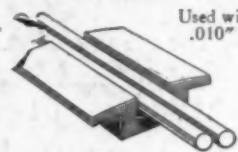
The slot, or channel, for the arch wire is not held to the close manufacturing tolerances found in the Edgewise Arch Bracket.



Used with .020" or .022" square wire



Used with .020" or .022" round wire



Used with two .010" wires

| Order No. | Pkg. of 10 | 10 Pkgs. of 10 | Pkg. of 250 |
|------------------|------------|----------------|-------------|
| M 464 .110" long | \$1.40 | \$13.50 | \$30.00 |
| M 465 .125" long | 1.45 | 14.00 | 30.00 |

BRACKET BANDS (Seamless and flat. All-precious metal)

| Order No. | Pkg. of 10 | Pkg. of 100 |
|---|------------|-------------|
| M 466X (M464 on flat band material $\frac{1}{8}" \times 1\frac{1}{4}" \times .004"$) | \$5.50 | \$49.50 |
| M 467X (M465 on flat band material $\frac{1}{8}" \times 1\frac{1}{4}" \times .004"$) | 5.50 | 49.50 |
| M 468X (M464 on flat band material $.150" \times 2" \times .004"$) | 6.50 | 58.50 |
| M 469X (M465 on flat band material $.150" \times 2" \times .004"$) | 6.50 | 58.50 |
| M 492X (M452† on seamless band No. 171) | 7.90 | 72.00 |
| M 493X (M464 on seamless band No. 171) | 6.70 | 60.00 |
| M 494X (M465 on seamless band No. 171) | 6.70 | 60.00 |
| M 495X (M452† on seamless band No. 170) | 7.30 | 66.50 |
| M 496X (M464 on seamless band No. 170) | 6.10 | 56.00 |
| M 497X (M465 on seamless band No. 170) | 6.10 | 56.00 |

*Available on special order. †M452 is Dr. E. H. Angle Edgewise Arch Bracket.

HALF-ROUND TUBES (All-precious metal)

| Order No. | Pkg. of 10 | Ounce or \$35.00 Rate | \$100.00 Rate |
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| M 62 .08" long | 2.50 | 2.425 | 2.00 |
| M 63 .09" long | | | |
| M 64 .10" long | | | |
| M 62Y } 1 dozen fitted on 2" length | 4.50 | 4.365 | 3.80 |
| M 63Y } shafting | | | |
| M 64Y } | | | |

Important: It is recommended that all half-round tubes be purchased fitted to the shafting to assure proper fit. They will be so supplied unless otherwise designated.

BUCCAL TUBES (All-precious metal)

| Order No. | Pkg. of 10 | Ounce or \$35.00 Rate | \$100.00 Rate |
|---------------------------------|------------|-----------------------|---------------|
| M 37 { .25" long for .036" wire | 3.00 | 2.91 | 2.80 |
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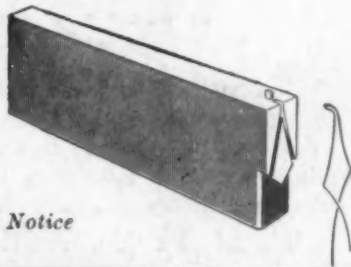
| Order No. | Pkg. of 10 | Pkg. of 100 |
|--------------------------------|------------|-------------|
| M 170 Seamless $\frac{3}{16}"$ | \$4.40 | \$40.50 |
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LIGATURE FORMER

For preforming ligature wire .007" to .010" for use with Brackets M 464, M 465, and the Edgewise Arch Bracket.

\$1.00 each

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